ETS World Conference

Macklowe Hotel & Conference Center
New York, New York

February 24-25, 1993

Professor Vincent T. Covello

School of Public Health
Columbia University
60 Haven Avenue, B-1, Room 106
New York, New York 10032
Telephone (212) 305-3464; Fax (212) 749-3590

Dr. Vincent T. Covello is a Professor of Environmental Sciences and Medicine, and Professor of Communications in the School of Public Health at Columbia University. He is also Director of Columbia University's Center for Risk Communication in New York City.

Prior to joining the faculty at Columbia, Dr. Covello was Director of Risk Assessment at the National Science Foundation, and a senior scientist at the White House Council on Environmental Quality in Washington, DC. He has also been a Study Director at the National Academy of Sciences, and a Professor at Brown University.

Dr. Covello has authored or edited over 25 books, and over 100 published articles on risk assessment, management, and communication. Among his most recent books are *Effective Risk Communication*, and *Risk Analysis: A Guide to Principles and Methods for Analyzing Health and Environmental Risks*.

Dr. Covello has lectured widely on risk communication topics, and has directed numerous training courses on risk communication for government agencies and corporations. In addition, he has authored risk communication guidelines and training materials for government agencies and corporations on risk analysis, risk communication, community relations, and related topics.

BIOSKETCH

Susan L. Santos

Ms. Santos is Research Program Director for Columbia University's Center for Risk Communication (CRC). As Research Program Director for the Center, Ms. Santos provides expert consultation in the areas of risk communication and assessment. Ms. Santos is Principal Investigator for several risk communication research programs and she also develops and implements a wide variety of risk communication training programs. Ms. Santos is also a part-time instructor in the Hazardous Materials Management M.S. Program at Tufts University. Ms. Santos received her undergraduate degree in Chemistry and Sociology at Boston College and her graduate degree in Civil Engineering and Public Health from Tufts University.

Ms. Santos has over fourteen years of environmental experience in the public and private sectors including 8 years at EPA Region I, primarily in the areas of toxic chemical and hazardous waste management. Ms. Santos has over six years of engineering consulting experience. Prior to her appointment at Columbia, Ms. Santos was Corporate Director for Risk Assessment Services with ABB Environmental. Ms. Santos' areas of expertise include the conduct and evaluation of human health and environmental risk assessments and risk communication. A specialist in the design, implementation, and evaluation of risk and environmental issue-oriented information and education programs, Ms. Santos has developed numerous information and communication strategies for hazardous waste sites, facility permitting, impact assessments, and community and worker right-to-know programs. Considered a leading risk communication practitioner, Ms. Santos has planned and participated as the lead communicator for close to 200 public meetings, hearings, citizen briefings and workshops on behalf of the government and industry. Ms. Santos has conducted Focus Groups, and direct mail survey programs as both research activities and as part of risk communication programs for industry and government agencies. Ms. Santos has also developed and served as the primary trainer/facilitator for over fifty Risk Assessment and Risk Communication training programs.

Ms. Santos has numerous publications in the areas of Risk Communication and Assessment. She has recently completed a report to the German Marshall Fund on a Comparative Study of Risk Assessment and Risk Communication Practices between Western Europe and the United States. This report is the product of a German Marshall Fund Fellowship Award to Ms. Santos in 1989.

Ms. Santos' interest in the communication of risk focuses on several issues including: how to facilitate two-way communication and decision-making among various players with different objectives; how to assist practitioners in understanding the role of risk perception in communication; how to explain risk information to various audiences and how to build trust and credibility. Recently, Ms. Santos has been focusing on risk assessment and risk communication strategies for Air Toxics, and the relationship of risk communication to pollution prevention activities. From an international perspective, Ms. Santos also works in understanding and facilitating cross cultural differences in risk communication.

HBI-USA
10378 Democracy Lane
Fairfax, Virginia 22030
(703) 352-0102
(703) 352-0151 (fax)

HBI Healthy Buildings International

Gray Robertson
President
Healthy Buildings International, Inc.
10378 Democracy Lane
Fairfax, Virginia 22030
(703) 352-0102

Gray Robertson is President and co-founder of Healthy Buildings International, Inc. (HBI) of Fairfax, Virginia. Born and raised in Liverpool, England, he graduated from London University in 1964 with a bachelor of science degree in chemistry and botany, including extensive study of mycology, or fungi.

Robertson spent three years as a bacteriologist with Evans Medical Company Ltd., Liverpool. He subsequently joined the Johnson Matthey Company, working for various subsidiaries in England and the U.S. as a chemist. He headed the chemicals division of the subsidiary, Matthey Bishop Inc., including supervision of its renowned analytical laboratories.

In 1980, Robertson began focusing on the problem of indoor pollution. In working in association with microbiologist/zoologist Peter Binnie, he helped to develop methodology to identify and eliminate the sources of indoor pollution.

In 1981, Robertson and Binnie co-founded HBI, a company devoted exclusively to the identification and control of internal pollution problems in public and commercial buildings. HBI has diagnosed problems and specified solutions in over 122 million square feet (11 million square meters) of occupied space in the United States, Canada, Britain, Switzerland, Finland, Sweden, Norway, Italy, Hong Kong, Venezuela, Australia and New Zealand. HBI now operates offices in Toronto, Canada; Sydney, Australia; Madrid, Spain; London, England; and the United States.

Developing a Risk Communication Strategy

Susan L. Santos

Increasing concern over environmental problems and the passage of more stringent legislation have led utility personnel to recognize that risk communication must become a part of their overall approach to environmental management. The key to effective risk communication is the two-way exchange of information between the utility and the public. The utility explains technical data to interested segments of the public, who in turn voice their concerns, opinions, and reactions. As well as being involved in a dialogue concerning the nature of the risk, the public should also be involved in decisions relating to its control.

Increasing concern over environmental problems, especially those involving toxic chemicals, has led to the realization that effective environmental management is not possible without effective communication. The complex decisions regarding the control of toxic chemicals in drinking water require more than just an understanding of the risks posed and the pollution-control mechanisms that are available. These decisions cannot be made independently by water utilities but require public involvement and risk communication.

Risk communication refers to the process of explaining or communicating environmental health and safety—or risk—information. In a 1986 report to the US Environmental Protection Agency (USEPA), Covello et al defined risk communication as "any purposeful exchange of information and interaction between interested parties regarding health, safety, or environmental risks."[1] This definition recognizes that risk communication, like any other form of communication, is a two-way process, involving a source that transmits a message via a communication channel, e.g., TV, radio, or newspaper, to a receiver. Defining risk communication in terms of basic communication theory illustrates how communication cannot occur without interaction between the source and the receiver.[2] Communication must be two-way. If this premise is accepted, then it is clear that risk communication is more than "explaining the data more clearly" or "explaining the numbers."

Risk communication helps explain technical information more clearly to lay audiences. Risk comparisons can be used that are not offensive or misleading, graphics can illustrate a point without obscuring the information, and the use of jargon in verbal presentations and written materials can be avoided. For it to be effective, however, risk communication involves much more than this. Risk communication is a process rather than a set of specific gimmicks or techniques. It requires awareness of the factors that affect the communication process and, perhaps more important, how individuals perceive risk and risk information. In fact, focusing on the communication *process* rather than just the *risk* may be one of the most important considerations for successful risk communication. In a nutshell, the key to effective risk communication is two-way communication.

Risk communication entails responding to the concerns, opinions, emotions, and reactions of the various stakeholders in the exchange.

Risk communication involves active listening, not just speaking. It entails responding to the concerns, opinions, emotions, and reactions of the various stakeholders in the risk-communication exchange and not just providing facts or responding to assumed misperceptions. Effective risk communication recognizes that the public has a right to receive information and to be actively involved in both the dialogue regarding the nature of the risk and in decisions about ways to minimize or control identified risks. This dialogue often blurs the distinction between risk assessment (Is there a risk? What is it and how bad is it?) and risk management (What should we do about the risk?). Issues of risk acceptability may become a part of the program even if there is no clear mechanism—or desire—to involve the public in these complex issues and decisions.

In developing a communication strategy, the focus should be on building strong communication channels between those who will communicate the information and the various audiences or receivers. This relationship needs to be built up over time, and it will change over time. It requires trust, openness, and honesty. Risk communicators must

be perceived as credible. If the source is not considered credible, the message—especially if it involves risk information—will not be believed. Although utilities may sincerely want to be considered good neighbors, this can only happen when management is committed to the idea that informing and involving the public are legitimate activities. Management must convey this commitment to all staff who may have the opportunity to interact with the public. Risk communication cannot be left to the official spokesperson. Risk communication is a part of everyone's job and is everyone's responsibility.

Developing an effective risk-communication program involves specific steps: (1) determining communication goals and objectives, (2) identifying the audience and its concerns, (3) understanding issues of risk perception that will influence the audience, (4) designing risk-communication messages and testing those messages, (5) selecting the proper communication channels, (6) implementing the plan, and (7) evaluating the risk-communication program.

Goals and objectives

Risk communication can have several goals and objectives. Sometimes the goal is to alert people to a particular risk and move them to action. For example, local authorities and utilities may be required to inform the public if testing shows contamination of the water supply or if certain chemical constituents exceed defined levels. At the other end of this spectrum, the public might have to be instructed not to drink the water or to boil it (in the case of some bacterial contamination) prior to consumption. At other times, the goal of risk communication is to tell people not to worry, to calm down. In these instances, the communicator wants to inform individuals that a particular situation does not pose a health risk. Utility managers find these situations particularly difficult. For example, how are testing results explained that show a drinking water constituent to be right below, right at, or right above the maximum contaminant level? Because people's concerns and information needs are different when they are being alerted and when they are being calmed down, strategies for communicating also need to vary.

Researchers have described some general purposes or objectives for risk communication:[3]

- education and information,
- improving public understanding,
- behavior change and protective action,
- organizationally mandated goals,
- legally mandated or process goals, and
- joint problem solving and conflict resolution.

An ideal goal might be for the public to better understand the technical issues and risk information and for the utility and government agencies to more fully appreciate the public's concerns, fears, and values. With the achievement of this objective, stakeholders can move from information and education to problem resolution. In reality, limitations of time, money, and personnel—and even regulatory constraints—may restrict the effectiveness of a risk-communication exchange.

Each event prompting the need for risk communication will have its own objectives. In designing a risk-communication program, the particular risk-communication needs and corresponding objectives are first determined. Goals and objectives lay the framework for the design of specific messages and activities. This framework establishes what needs to be communicated and why.

A 1986 report by the USEPA described four main rules for risk communicators:[1]

- know your risk-communication problem,
- know your risk-communication objective,
- use simple and nontechnical language, and
- listen to your audience and know its concerns.

Identifying audience, concerns

Often, risk communicators, and in particular public agency officials, inadvertently place too much emphasis on the design of a particular risk-communication message and how to simplify or better explain the technical risk information. To ensure that communication is two-way, more attention should be focused on the receiver. This means first identifying the various audiences.

Although it may not be possible to reach everybody, it is important to try to identify individuals and groups who have an interest or stake in the issue and to provide an opportunity for these people to be involved. As a starting point, existing mailing lists or geographic information on member communities may be used. Within a particular geographic area, several tiers of audiences will exist that may include individuals or groups with a particular interest in the issue. Your audience, however, should not be limited to just geographic neighbors. Other audiences may exist based on common demographic, educational, or other interests (see side bar).

When identifying audiences, it is important to recognize that individuals and groups will frequently have concerns and information that they wish to share. Thus, audience identification goes beyond just determining who needs to be informed. The fundamental challenge of risk communication is what to do with the people who have something to say. Their concerns, of course, must be documented. This can be thought of as a data-collection activity—as well as an exercise in active listening. For effective

Checklist to aid in audience identification

- Local government agencies, e.g., (board of health, planning commissions)
- Education groups
- Academic institutions
- Local elected officials
- State and federal government agencies
- Chambers of Commerce
- Unions
- Professional organizations
- Local, regional, and national environmental groups
- Local businesses
- Civic organizations
- Community associations
- Property owners
- Religious organizations
- Senior citizen associations
- Public interest groups
- Sporting and recreational clubs
- Media
- Other interest groups

communication to occur, public concerns must be known prior to the relaying of risk information. Only then can the message be presented and disseminated in a manner that acknowledges and addresses the attitudes and apprehensions of the receivers.[2]

Although audience concerns vary from situation to situation, it is possible to categorize them. Chess et al[4] developed four general categories of concerns: (1) health and lifestyle concerns, (2) data and information concerns, (3) process concerns, and (4) risk-management concerns. Health and lifestyle concerns are often the most important because in any risk situation, people inevitably want to know what the implications are for themselves and their families. Santos and Edwards[2] described this as the "What does it mean to me?" series of questions. Citizens want to know if they will be "safe." For water utilities, this translates to any number of specific concerns, such as: Can I drink the water? Will I get cancer? Can I use the water to cook or to irrigate my garden? Will my water rates go up? Because these are commonly the first questions people ask, risk-communication messages must clearly address and respond to these issues.

Data and information concerns are usually associated with the technical basis for—and uncertainties involved in—any estimation of risk, e.g., Are your studies correct? Did you sample for the right parameters? Process concerns relate to how decisions are made by the utility or responsible agency when responding to a risk and to how communication occurs: Who decides? How are we informed? Obviously, trust and credibility are important in these issues, as is the control the public feels it has in the decision-making process. Finally, risk-management concerns relate to how and when the risk will be handled; i.e., Will it be effectively mitigated, avoided, or reduced? To answer this concern, citizens will often look to an organization's previous track record in making decisions and responding to risk situations.

A variety of techniques is available for documenting audience information needs and concerns, including interviews, written or telephone surveys, information gathering (e.g., shopping mall exit interviews), the use of existing public poll information, review of news coverage and letters to the editor, small informal community group meetings, and focus groups. Focus groups are being used more and more as a relatively inexpensive means of identifying audience attitudes, opinions, and concerns. Participants are selected from a specific target group or from the general population. Besides being employed to identify audience concerns, focus groups can be effectively used to test risk messages. When carefully designed and conducted by an experienced facilitator, focus groups can serve as a valuable source of information.

A utility's trustworthiness can be increased by collaboration with credible sources who can help to communicate the utility's message to the public.

Once audiences have been identified and their needs and concerns have been determined, the information is rated as to its relative importance. Because risk-communication resources are limited, the emphasis should be on key issues and the most important audiences.

Risk perception

In addition to listening effectively, utilities must try to understand how the public perceives risk and risk information. Frequently, the public views a risk quite differently from the way a public agency or utility views the risk. Various researchers have identified and classified more than 25 characteristics of risk perception.[5,6] Sandman[6,7] coined the term "outrage" to describe a variety of factors that influence the public's perception of risk. Outrage, as defined by Sandman, is everything about a risk *except* how likely it is to cause harm. To truly understand audience needs and concerns, risk communicators should analyze a particular situation to see what outrage factors are at play. These risk-perception issues can dramatically influence communication exchange. Sandman's categorization of risk perception characteristics as outrage factors depends on whether the risk is perceived as

- voluntary or involuntary,
- controlled by the system or controlled by the individual,
- fair or unfair,
- having trustworthy or untrustworthy sources,
- morally relevant or morally neutral,
- natural or artificial,
- exotic or familiar,
- memorable or not memorable,
- certainty or uncertainty,
- undetectable or detectable, and
- dreaded or not dreaded.

Voluntary or involuntary. Risks that are voluntary are usually perceived by the public as less serious, i.e., less dangerous, than those that seem to be involuntary or imposed. When people feel that a risk is being imposed on them, they perceive it as outrageous and they attribute to it a higher level of risk—regardless of the actual hazard. A voluntary risk (such as smoking or driving without buckling the seatbelt) should never be compared with a perceived involuntary risk (such as drinking contaminated water). To make such a comparison would greatly heighten citizen outrage.

Controlled by the system or the individual. People tend to view risks that they cannot control as more threatening than those that they can control, regardless of the actual hazard. Water contamination and concentrations of toxic pollutants (whether regulations deem them allowable or not) are perceived to be beyond the control of the individual. In the area of drinking water, in particular, outrage will increase if the public feels that utilities or local government agencies have all the control over the perceived risk. In order to deal with this factor, the utility or agency can establish a mechanism through which citizens have a voice in the decision-making that relates to the control of contaminants or other water supply issues that affect their lives.

Trustworthy or untrustworthy sources. How individuals view a risk is often a function of how much they trust the organization that seems to be imposing or allowing the risk and of how credible they believe the source of risk information is. A utility's trustworthiness and credibility can be increased by its collaboration with credible sources outside the organization who can help to communicate the utility's message to the public. Trust and credibility have to be nurtured; they don't just happen.

Morally relevant or morally neutral. Risks that are ethically objectionable will be perceived as more dangerous than those that are not. Many people feel that pollution is morally wrong, and this makes talk of cost-risk tradeoffs sound callous. This feeling contributes to the public's desire to reach a zero-risk level. It is important that utilities acknowledge that pollution or contamination at any level is wrong—which is not the same as saying it will cause a health problem.

Recognizing and acknowledging feelings of this kind will aid a utility in gaining the acceptance of realistic cleanup goals. It also is important to note that there was not malicious intent to create the risk, but there is committed intent to mitigate the risk to the greatest extent technology permits.

Exotic or familiar. Exotic risks appear more risky than familiar risks. For example, household cleaners seem less risky than the chemical plant that makes them. Toxic pollutants, with their long names, can certainly seem exotic. The challenge for risk communicators is to try to remove some of the mystery surrounding these pollutants and their effects. Demystifying the risk will help get the message across.

Dreaded or not dreaded. Risks that are dreaded seem more serious than those that carry less dread. For example, toxic chemicals that are carcinogens may seem more risky and less acceptable than those that cause emphysema, even though both are capable of causing diseases that can be fatal. It is important that communication efforts recognize and acknowledge this dread. Health effects associated with acute exposure must be differentiated from those associated with chronic exposure and carcinogenic effects differentiated from noncarcinogenic effects. The risk message puts the health effects information into the proper perspective, so that people can comprehend the difference between significant and less significant risks. This can be especially important for risks associated with drinking water.

Certainty or uncertainty. Risks that are thought to be more certain or known are often perceived by the public to be less serious (and more acceptable) than those that are not. Conversely, risks that scientists are uncertain about are considered far more serious. In these cases, the public tends to want to err on the side of caution; that is, it does not want to accept risks that are uncertain. Therefore, risk-communication efforts must acknowledge points of uncertainty to maintain credibility.

Risk-perception considerations cannot be ignored or minimized as emotional, unfactual, or irrelevant. Emotions, feelings, values, and attitudes carry as much—if not more—importance for the public than the technical magnitude of the risk situation. Utilities must recognize and acknowledge that risk perception is not public hysteria. An appropriate starting point for potential risk communicators might be to determine the risk perception factors at work and how they might affect communication.

Many risk analysts tend to use overly technical and bureaucratic language, which is not appropriate for communicating with the general public.

Designing messages

As well as responding to citizen concerns, a risk communication message includes information that the utility wants to relay, such as facts about

- the organization and its credibility,
- the project,
- the reasons for undertaking the project,
- the risks or impacts,
- the precautions that have been built into the plan,
- the unknowns and how the organization intends to account for these uncertainties, and
- the involvement of the public.

After the content of the message has been defined, the next step is deciding how to state or present it. Will the message be written or presented orally? If presented orally, who will be the communicator?

Written messages and oral presentations must transmit the information to the public in an understandable form. Many risk analysts tend to use overly technical or bureaucratic language, which may be appropriate for the risk-assessment document and for discussions with other experts but not for communicating with the general public. Scientists are by nature precise and, as such, tend to describe all the uncertainties and limitations associated with a risk assessment. This is frightening and overwhelming for the public, who is trying to figure out what the risk means and wants certainty—not caveats. Rather, the message should explain, as simply and directly as possible, such things as risk estimates, exposure considerations, and risk comparisons. Because different audiences have different concerns and levels of understanding, one risk message may not be appropriate for all interested parties. It may be necessary to develop a series of messages on the same topic.

A good communicator is equally comfortable in active listening and in public speaking. Training technical and management staff to become effective communicators should be a key part of a utility's overall risk-communication strategy.

A critical part of successful message design is testing or trying out the message. This can be done formally, e.g., by the use of focus groups or citizen advisory committees, or informally, e.g., by testing the material on uninformed third parties.

In the process of testing the message, the utility is looking for feedback. Is it clear? Does it meet objectives? Will it evoke outrage? Does it answer overt questions? Does it address underlying concerns? Once these questions have been answered, the script can be revised and the message finalized.

Selecting communication channels

Next, the best way to "get the message out" has to be determined. The challenge is to find the right channel of communication for each audience, because one channel may not be appropriate for all. Also, the choice of channels may change over time, i.e., at different project phases such as study, design, implementation, and crisis response.

Communication channels are selected that make the best use of resources while still meeting overall goals. For example, door-to-door evening visits with neighbors might be identified as the best way to reach a target audience, but staff may not be available for conducting such visits. Instead, the organization might opt for a newsletter, hand-delivered by employees, or decide on several informal community meetings in citizens' homes.

Techniques for communicating with the public include the use of

- brochures,
- information packets,
- newsletters (perhaps one that is published regularly),
- videotapes or slide shows,
- advertisements,
- fact sheets, and
- press releases.

Plant activities might include

- open house and plant tours,
- emergency drills and exercises, and
- educational and informational workshops.

Other outreach activities include communicating through

- community meetings,
- community advisory groups,
- service group presentations,
- educational activities with schools,
- the news media (e.g., radio and television interviews),

- other credible sources or existing groups, and
- a telephone hotline established by the organization.

It's important to target outreach activities to meet the needs of specific audiences.

Public meetings, especially formal ones, are a common communication technique. Thorough planning for these meetings—from logistics to determining the meeting agenda and objectives—is critical. Some general issues also need to be considered, e.g., providing background information and ensuring that the audience understands and, therefore, receives the information you want to communicate. In an ideal situation, educational information is distributed prior to the meeting at which results are being explained. It may also be appropriate to provide materials that people can take home, such as fact sheets summarizing drinking water analyses, the risk assessment, or the water treatment process.

For any risk-communication activity—particularly for public meetings—a utility can consider collaborating with other credible sources, such as scientists, doctors, and educators. If the subject to be discussed is controversial, thought should be given to having an impartial moderator run the meeting. A successful meeting is frequently one that has been structured to give as much control as possible to the public—while still being conducted in an orderly fashion.

Just as written materials need to be tested, public meetings need to be rehearsed in anticipation of tough questions that may arise.

Evaluating the program

The last step is an evaluation of the implemented risk-communication program. At a minimum this involves evaluating previous communication outreach activities, such as public meetings; evaluating written materials, such as fact sheets and brochures; and evaluating media interaction and news coverage. Kline et al[8] suggested several components for an evaluation program.

Audience analysis. Audience analysis is designed to help agencies understand audience perceptions and solicit feedback from key audiences before, during, and after a communication program. Such analyses can include discussions by utility staff of predicted audience positions, gathering questions from audiences in advance of meetings, analyzing news clippings, public opinion polling, and qualitative questionnaires.

Message pretesting. Such pretesting allows agencies to gather feedback on written materials before they are printed and distributed. Message pretesting can include surveys and questionnaires; discussion groups, e.g., focus groups; and reviews of the language used in the materials.

Assessing communicator style. These assessments help organization spokespersons realize what attitudes and strengths they bring to risk communication. In the past, risk communicators have, typically, focused on scientific facts, a focus that may not be the same as the audience's and can lead to an impasse in communication. Most of the tools available to assess communicator style are self-assessment surveys that are completed and then scored, providing a profile of the respondent's style and motivational pattern.

Outcome analysis. Outcome analysis tools, which examine communicator performance and audience reaction, include meeting reaction forms for participants in public meetings, verbal feedback, speech evaluation checklists, and internal observation and debriefing. Basically, these tools help answer the questions: How did we do? and What could we do differently?

Once evaluation is complete, it is time to review the risk-communication program and make any necessary adjustments. The tendency to delay thinking about communication until the next crisis should be avoided. Commitment to an ongoing risk-communication program may not have dramatic results at first but, with time, the benefits will be evident.

Summary

Risk communication is still a relatively new concept, but it is gaining much attention. Organizations—public and private alike—are realizing that communication is part of an evolving culture. Such cultures recognize not only that the public is demanding more information and is entitled to information that affects it but also that risk communication can improve the risk-management decision-making process and directly benefit company operations. With the passage of more stringent environmental legislation, including an increasing number of drinking-water regulations, risk communication must become a part of a utility's overall approach to environmental management. Advance planning will facilitate the risk-communication process and set the stage for effective and meaningful two-way communication.

References

1. COVELLO, V.T.; VON WINTERFELDT, D.; & SLOVIC, P. Risk Communication: An Assessment of the Literature on Communicating Information About Health, Safety and Environmental Risks. Draft Preliminary Rept. to USEPA (Jan. 1986).
2. SANTOS, S.L. & EDWARDS, S. Unravelling Risk Communication. APHA Conf., New Orleans, La., Oct. 1987.
3. KEENEY, R.L. & VON WINTERFELDT, D. Improving Risk Communication. *Risk Analysis*, 6:4 (1986).
4. CHESS, C.; HANCE, B.J.; & SANDMAN, P. *Planning Dialogue with Communities: Risk Communication Workbook*. Envir. Communication Res. Program, Rutgers Univ., New Brunswick, N.J. (June 1989).
5. SLOVIC, P. Perception of Risk. *Science*, 236:280 (1987).
6. SANDMAN, P. Explaining Environmental Risks—Some Notes on Environmental Risk Communication. USEPA (Nov. 1986).
7. CHESS, C.; HANCE, B.J.; & SANDMAN, P. *Improving Dialogue With Communities: A Risk Communication Manual for Government*. Envir. Communication Res. Program, Rutgers Univ., New Brunswick, N.J. (1988).
8. KLINE, M.; CHESS, C.; & SANDMAN, P. *Evaluating Risk Communication Programs: A Catalogue of "Quick and Easy" Feedback Methods*. Envir. Communication Res. Program, Rutgers Univ., New Brunswick, N.J. (May 1989).

About the author: *Susan L. Santos is Director of Corporate Risk Assessment Services, ABB Environmental Services Inc., 107 Audubon Rd., Corporate Place 128, Wakefield, MA 01880, and also holds an appointment to Columbia University's Center for Risk Communication in New York. As research program director for the center, Santos provides consultation in the areas of risk communication and assessment, and she develops and implements a variety of risk-communication training programs. She is also a part-time instructor in the hazardous materials management program at Tufts University in Medford, Mass. Santos is a graduate of Boston College (Chestnut Hill, Mass.) with a BS degree and of Tufts University with an MS degree. Santos' work has been published previously by* Environmental Science & Technology *and* Journal of Facilities Management Operations and Engineering—AIPE.

Tobacco Control 1992; 1: 208–219

INDUSTRY WATCH

Issues and answers concerning passive smoking in the workplace: rebutting tobacco industry arguments

James L Repace, Alfred H Lowrey

Abstract

In response to increased demands for smoke-free workplaces, the multinational tobacco industry has mounted a worldwide public relations campaign to create the appearance of scientific controversy over the health effects and control of environmental tobacco smoke (ETS). We rebut allegations by the tobacco industry and its apologists concerning ETS. We present arguments that show that ETS does cause disease; that ETS is significant relative to other indoor pollutants; that current atmospheric and biological markers for ETS are appropriate; that non-smokers are exposed to sufficient amounts of ETS in workplaces to cause disease; that non-smokers' exposures to ETS have been properly assessed in epidemiological studies; that there is indeed a scientific consensus about the health effects of ETS; and that ventilation or other measures short of eliminating non-smokers' exposures are inadequate controls for ETS.

(*Tobacco Control* 1992; 1: 208–19)

Introduction

In the United States and other countries there is public demand for restrictions on environmental tobacco smoke (ETS) in the workplace. US surveys show that in 1987, 90% of lifelong non-smokers and 89% of ex-smokers as well as 80% of current smokers were in favour of restrictions on smoking in workplaces.[1] In the United States, however, the occupational health authorities with regulatory authority over workplace air quality have only recently moved tentatively towards regulating smoking at work.[2]

The tobacco industry has argued that restrictions on smoking indoors are unnecessary. It argues, for example, that data on the health effects of ETS are inconclusive; that non-smokers are exposed to trivial amounts of tobacco smoke compared with smokers; that other indoor pollutants are far more important than ETS; and that solutions other than stringent workplace policies should be pursued – for example, using "common courtesy" to accommodate the needs of both smokers and non-smokers, providing designated smoking areas on the same ventilation system, and improving engineering controls such as ventilation or air cleaning. Because regulatory officials and employers unfamiliar with advances in ETS research may possess inadequate information on such topics, we present specific rebuttals to arguments abstracted from publications, advertising copy, or other statements by the tobacco industry and its allies in the public domain.[3,4]

(1) "Common courtesy controls passive smoking"

The tobacco industry suggests that non-smokers bothered by tobacco smoke should "mention annoyances [from ETS] in a pleasant and friendly manner," and that smokers, before lighting up, should tender "the ancient courtesy of 'Do you mind if I smoke?'"[5]

REBUTTAL

This in effect requires concerned non-smokers and smokers to canvass all those present in a space and inform them of their preferences or intentions. Thus it is not surprising that when Davis *et al* analysed the results of the 1987 National Health Interview Survey of 22000 US adults to determine whether common courtesy was being used in passive smoking situations, they found that 47% of the smokers reported lighting up inside public places without asking if others mind, while only 4% of non-smokers asked a smoker to refrain.[6] Davis *et al* concluded that these findings show that the common courtesy approach is unlikely to eliminate non-smokers' exposure to ETS.[6]

(2) "Complete elimination of ETS is unnecessary"

The tobacco industry argues that the premise that workplace exposures to ETS must be eliminated is without adequate supporting data.[7]

REBUTTAL

This argument is equivalent to saying it should be presumed that thresholds (defining safe and unsafe levels of exposure) exist for all the diseases of smoking and that all non-smokers have exposures beneath their individual

101 Felicia Lane, Bowie, Maryland 20720, USA
J L Repace

7016 Forbes Boulevard, Seabrook, Maryland 20706, USA
A H Lowrey

Correspondence to: Mr Repace.

Mr Repace, a physicist and an expert on indoor pollution, was a 1989 recipient of the US Surgeon General's Medallion for his work on passive smoking. Dr Lowrey is a research chemist – ED

thresholds. The argument runs directly counter to the advice of public health agencies. Given the public health information on the massive harm that smoking inflicts, would such an argument be persuasive if the tobacco industry proposed to add tobacco smoke condensate as an additive to food, beverages, or cosmetics? The US Public Health Service has stated that spacial separation of smokers from non-smokers may reduce, but not eliminate, non-smokers' exposure. In recent public health advisories US public health agencies such as the National Institute for Occupational Safety and Health[9] and the US Environmental Protection Agency[8] have proposed smoke-free workplaces or separately ventilated smoking areas, because even low level exposures to carcinogenic agents are presumed to carry an increased risk of cancer. Other options, such as unrestricted smoking, designated smoking and no-smoking areas in a shared space or on the same ventilation system, or increased ventilation and air cleaning, do not eliminate non-smokers' exposures.[10]

(3) "ETS is far less important than other indoor pollutants"

The tobacco industry argues that, by focusing on ETS, public health agencies are diverting attention away from more serious workplace problems such as electromagnetic fields, volatile organic compounds, and microbes. They assert that the sick building syndrome, formaldehyde, sulphur oxides, ammonia, oxides of nitrogen, and ozone cause symptoms identical to ETS and that public health authorities have unfairly singled out tobacco smoke, ignoring many "real" threats to public health and safety.[7,11,12] (Tobacco industry-sponsored magazine articles aimed at smokers' rights groups have even cited the alleged higher-than-ETS lung cancer risks due to "owning a bird" or "drinking green tea or pasteurised milk.")

REBUTTAL

ETS risks are among the most serious airborne environmental pollution hazards ever considered by public health agencies. For example, research at the US Environmental Protection Agency has shown that smoking is the largest source of particulate indoor air pollution and is also the major combustion source contributing to total human exposure to mutagens and carcinogens.[13] In addition, several epidemiological studies have reported the occurrence of health effects when non-smokers breathe ETS at environmental concentrations (so called passive or involuntary smoking). The 1986 US Surgeon General's report stated that a comparison of the chemical composition of the smoke inhaled by active smokers with that inhaled by involuntary smokers suggests that the toxic and carcinogenic effects are qualitatively similar, a result of the fact that both types of smoke derive from tobacco combustion.[14] Although several epidemiological studies report carcinogenic effects of electromagnetic forces with the same odds ratios as for ETS, a comparison of the odds ratios in epidemiological studies of the risks of ETS and electromagnetic forces is erroneous and highly misleading because the denominators of those ratios pertain to different control groups. Moreover, there is no comparable body of published work implicating electromagnetic forces as a massive cause of cancer at high exposures and little evidence of dose-response relations. Thus, the weight of evidence against ETS is incomparably stronger than that against electromagnetic fields. To place the relative contribution of ETS as a pollutant into perspective, the impact of ETS on human mortality has been estimated to be two orders of magnitude greater than that of all regulated hazardous outdoor air pollutants combined.[15]

It is also important to note that the symptoms of the sick building syndrome – headaches, dizziness, nausea, and eye, nose, and throat irritation – are also caused by ETS. The irritant chemicals acrolein, acetaldehyde, formaldehyde, sulphur and nitrogen oxides, and ammonia, as well as many other irritating volatile organic compounds, are present in tobacco smoke. The known irritant effects of ETS are indistinguishable from other unknown causes of sick building symptoms.

(4) "There is little evidence, and nothing which proves scientifically, that cigarette smoke causes disease in non-smokers."[16]

REBUTTAL

This widely advertised statement was challenged as false advertising by health groups in the Australian Federal Court, and after extensive evidentiary hearings a ruling was handed down that scientific evidence does indeed establish cause and effect between passive smoking and lung cancer, as well as asthma and respiratory problems in children. The court enjoined the Australian tobacco industry from continuing to advertise its false claims.[16]

Thru The Smoke Screen, published by the New Zealand Ministry of Health, observes that tobacco industry pronouncements on ETS are deficient in three main areas.[17]

(1) Documents cited by the tobacco industry do not distinguish between refereed journal publications and unrefereed offerings such as letters to the editor; the industry often cites reports of offerings in industry-sponsored symposia or conferences that have not undergone scientific peer review and have less standing in scientific circles.

(2) The tobacco industry chooses published work selectively, ignoring most of the recent work showing the relation between ETS and disease or death.

(3) The tobacco industry misquotes or quotes out of context statements of writers whose work they review. For example, on numerous occasions the tobacco industry has quoted out of context this sentence from an IARC (International Agency for Research on

Cancer) monograph on tobacco smoke: "The observations on non-smokers that have been made so far are compatible with either an increased risk from passive smoking or an absence of risk."[16] Always omitted, however, is the *following* sentence, which states in part: "Knowledge of the nature of sidestream and mainstream smoke, of the materials absorbed ... and of the quantitative relationships ... commonly observed from exposure to carcinogens, however, leads to the conclusion that passive smoking gives rise to some risk of cancer."[16] Reinken further indicts the tobacco industry for its personal attacks on individual researchers (see, for example, *Science* 1987 236: 250–1), for its attacks on standard methods of statistical analysis, for focusing on small and inconclusive studies while excluding large well done ones, and for misrepresenting scientific evidence.[17]

Other authors have also observed that the tobacco industry is engaging in an elaborate international campaign to refute the evidence on the harm of passive smoking for three main reasons. Firstly, the passive smoking issue allows a widening of the definition of smoking beyond that of a purely personal behaviour, legitimising it as a social problem. Secondly, successful cases of litigation against employers by workers with histories of long term exposure to ETS have created an industrial climate of concern leading to workplace smoking restrictions and bans. Thirdly, the proliferation of smoking restrictions reduces smoking opportunities and thus reduces total cigarette consumption and hence industry profits.[19] For example, a survey of smoking rates was made in a Canadian federal workplace by Health and Welfare Canada before and after smoking restrictions. The number of cigarettes smoked at work was reduced by 29% as a result of restrictions.[20] Stillman *et al* reported up to a 20% reduction following smoking bans in a hospital in the United States.[21]

(5) Existing epidemiological studies of workplace exposures to ETS do not provide adequate support for restrictions against smoking in the workplace[11]

REBUTTAL

Epidemiological studies of passive smoking show that smoking by their spouse is a cause of lung cancer in non-smoking women. Clearly, if exposure to passive smoking at home causes lung cancer, exposure in other places must also carry an increased risk. Several clinical studies have indicated that the workplace is an important source of exposure to ETS. For example, in an international study of urinary cotinine levels in 1369 non-smoking women from 10 countries, Riboli *et al* found a clear linear increase from the group of women exposed neither at home nor at work to the group of women exposed both at home and at work.[22] In a similar study of urinary cotinine values in 663 male and female lifelong non-smokers and ex-smokers in New York State in 1986 Cummings *et al* found that exposure to ETS is extremely prevalent, even among those not living with a smoker; the most frequently mentioned sources of exposure were at work and at home.[23] In another New York State study of 380 lifelong non-smokers a total of 87% of subjects reported exposure to ETS at work.[24] Moreover, time budget studies show that the most-frequented microenvironments are the home and the workplace.[25]

Smaller epidemiological studies of passive smoking in the workplace generally have not found a lung cancer effect; however, the world's largest case-control study of passive smoking and lung cancer to date (over 400 cases and 800 controls) has found that whereas smoking by a spouse was associated with a 21% increased risk of lung cancer (95% confidence interval 0·96 to 1·54), passive smoking in the workplace was associated with a 34% increased risk of lung cancer (1·03 to 1·73), and when adenocarcinoma alone was considered, the lung cancer risks associated with both types of exposure increased to about 40% and were statistically significant.[26]

White and Froeb, in studying the effect of long term workplace passive smoking in 2100 middle aged workers, found that workers who were exposed at work suffered significant declines in pulmonary function (indicative of small airways dysfunction) relative to those who were not exposed at home or at work.[27] A more recent study by Masi *et al* found that the vital capacity and total lung capacity of men, and the lung diffusing capacity in women, were significantly adversely affected by passive smoking in the workplace.[28]

(6) Non-smoking workers are not irritated by ETS in the workplace; ETS exposures are below the threshold of discomfort[29]

REBUTTAL

A question on discomfort from cigarette smoke in the workplace was included in the Occupational Health Supplement to the 1988 National Health Interview Survey. This was a cross sectional household interview survey of approximately 44000 adults (aged 18 or more representative of the US civilian, non-institutionalised population. Among employed non-smokers who reported that their workplace was not in their home, 36·5% (or 28·5 million Americans) worked at places that permitted smoking in designated (if any) and other areas. Of these, 43·5% (12·4 million) reported some or moderate discomfort and 15·7% (4·5 million) reported great discomfort from ETS at the workplace.[30]

Worker discomfort from workplace passive smoking has also been documented by Barad. Labour-management strife over workplace smoking at the Social Security Administration (SSA) in Baltimore, Maryland, United States, led to a study of 10000 non-smoking workers. Barad found that passive smoking impaired the work efficiency of more than half of the workers.[31] Thirty-six per cent of the workers reported that they were forced to move away from their workstations to avoid breathing

smoke, 21%, reported difficulty in concentrating on work because of passive smoking, and 14%, found it difficult to produce work as a result of ETS in the workplace. Frustrated by the lack of resolution by management of its complaints about passive smoking, a group of non-smoking workers filed a suit against the management and pursued the case to the US Supreme Court. The court subsequently dismissed the lawsuit for lack of jurisdiction.[32]

(7) The US National Institute for Occupational Safety and Health has shown that ETS is a sick building problem in only 2–3% of commercial buildings[29]

REBUTTAL

Although the National Institute for Occupational Safety and Health (NIOSH) has in the past reported that only 2% of its sick building investigations have dealt with tobacco smoke,[33] this is misleading. Firstly, although the symptoms of ETS exposure and the sick building syndrome are similar, NIOSH investigators are not likely to be called in when workers are reacting to symptoms which they attribute to environmental tobacco smoke exposure.[31,34] When the cause of complaints is known, it generally becomes a labour-management dispute, resulting in arbitration or litigation.[32,34] In presentations at indoor air quality seminars we have witnessed NIOSH staff identifying ETS as a significant indoor air pollutant. Secondly, NIOSH staff have stated that the institute currently has no standard method of measuring ETS and has relied upon carbon monoxide measurements as a surrogate. In general, carbon monoxide measurements are not a good indicator for ETS. NIOSH has stated that "the actual role of ETS in relation to building occupant complaints in [NIOSH's] indoor air quality studies has not yet been objectively determined by NIOSH researchers" (K Wallingford, 1991, written communication). Even more importantly, NIOSH recently published an advisory declaring ETS to be a "potential occupational carcinogen" (using legal terminology of the US Occupational Safety and Health Administration [OSHA]) and recommending elimination of tobacco use from the workplace.[3] OSHA has published a *Federal Register* notice stating that "secondary tobacco smoke is a recognized health hazard which OSHA is considering for separate regulatory action."[3]

(8) Smokers should have freedom of choice to smoke[35]

REBUTTAL

Smokers should not have freedom of choice to liberate toxic and carcinogenic chemicals in the breathing zone of non-smokers. Smokers can choose to refrain from smoking indoors, can choose to smoke outdoors, or can choose to stop. Non-smokers cannot choose to refrain from breathing. Smokers' pollution harms non-smokers; non-smokers' breathing does not harm smokers. *Smokers are on the same moral* ground as were spitters around the turn of the century, when public health laws restricted tobacco chewers from spitting on the floor of public buildings.[32]

(9) Components of ETS, even if toxic in other settings, have not been proved to be toxic in ETS

REBUTTAL

Levels of ETS-generated respirable suspended particulates (see argument 10 below) in buildings typically exceed the levels of the National Ambient Air Quality Standards.[36] Compared with smokers, non-smokers experience low dose exposures to sidestream smoke, exhaled mainstream smoke, and ETS. Smokers are exposed to higher doses of these smoke fractions plus very high doses of mainstream smoke. More than 4700 compounds have been identified in tobacco smoke, of which fewer than 500 are in the gas phase.[36] At least 43 of these compounds are known human or animal carcinogens.[18,37] As discussed in the rebuttal to argument 2, there are no known thresholds for carcinogenesis (also see rebuttal to argument 21).

Quantitative comparisons have been made of the mainstream and sidestream smoke of four types of popular US cigarettes.[38] The analyses generally show that toxic and carcinogenic agents studied are higher in sidestream smoke than in mainstream smoke, and that the carcinogenic potential of indoor pollutants resulting even from low yield cigarettes is not diminished.[38]

Furthermore coexposure to a variety of environmental agents may increase the risk from tobacco smoke: Exposure to radon daughters seems to interact synergistically with tobacco smoke in increasing the risk of lung cancer.[39] The increased lung cancer risk from coexposure to pulmonary disease-producing occupational dusts such as coal and silica dusts, for example, is additive to that produced by tobacco smoke. Exposure to asbestos produces a synergistic reaction with tobacco smoke for both asbestosis and lung cancer. Other industrial toxicants such as petrochemicals, aromatic amines, and pesticides are also suspected to interact with tobacco smoke exposure and increase disease risk.[40]

(10) Assessment of non-smokers' exposure to ETS is inadequate for risk assessment, and based on outdated studies, questionnaires, or models whose utility in predicting ETS exposures is exaggerated.[7,11,12] ETS exposure cannot be assessed using currently available atmospheric markers such as respirable suspended particulate matter (RSP) or nicotine. ETS-derived RSP levels are not a major contributor to workplace air pollution.[41]

REBUTTAL

ETS is well established as a major contributor to indoor air pollution, including that of workplaces.[34] Current studies, questionnaires, and models have shown an ability to predict ETS exposures reasonably well.[42-44] Models show that ETS concentrations are directly proportional to the smoker density (number of smokers per volume of space) and inversely proportional to the rate of air exchange. In general, if the number of smokers in the space is greater than two, on average, a steady stream of smoke is generated.[45] A number of studies have shown that non-smokers' ETS exposures are determined by the product of the ETS concentration, the duration of exposure, and the respiration rate during exposure.[46] Based upon urinary cotinine studies, Riboli *et al* have shown that, when appropriately questioned, non-smoking women (upon whom most epidemiological studies of passive smoking are based) can provide a reasonably accurate description of ETS exposure, and that both the duration of exposure and number of cigarettes to which subjects reported being exposed were strongly related to urinary cotinine values.[47]

The significant contribution of ETS to RSP levels indoors has been reported by both the US Surgeon General[14] and the National Research Council (NRC).[25] The NRC's report concluded that a majority of field studies have used RSP as an indicator of exposure to ETS because of the "substantial emission of RSP in indoor spaces from tobacco combustion." The NRC also stated that "Total RSP, as measured by personal monitors, has been found to be substantially elevated for individuals who report being exposed to ETS as compared with those who report no exposure," and that "Both air monitoring and modeling clearly indicate that RSP concentrations will be elevated over background levels in indoor spaces when even low smoking rates occur."[25] The Surgeon General concluded that "It has been demonstrated that ETS has resulted in elevated respirable suspended particulate levels in enclosed spaces."[14]

Insofar as the contribution of ETS to workplace exposures is concerned, a review of recent work has shown that ETS remains a significant workplace contributor to indoor air pollution. We describe three examples.

(1) Nelson *et al*[48] measured aerosol mass concentration continuously over five 24 hour periods in a 700 m^3 (25000 ft^2 floor area) office with one smoker (daytime occupancy, smoking rate not reported) and an air exchange rate of 1 air change per hour[49]; the large impact on office air caused by smoking is apparent by comparing the daytime office aerosol concentrations (of the order of 50 $\mu g/m^3$) during smoking occupancy and evening RSP concentrations (of the order of 18 $\mu g/m^3$) during its absence. In modelling the concentration for such an office area, Repace predicted a value for a chain-smoker (smoking at a rate of 6 cigarettes per hour in a volume of 700 m^3 at an air exchange rate of 1 per hour, with an 18 $\mu g \cdot m^{-3}$ background added of 48 $\mu g/m^3$, consistent with observations.[45]

(2) Nagda *et al* measured concentrations of RSP in the smoking section of a random sample of 69 smoking and 23 non-smoking flights for the US Department of Transportation.[17] Non-smoking flight attendants must work in the smoking sections on aircraft where smoking is still permitted – for example, for the United States on most international flights. Levels of RSP on the smoking flights averaged 175 $\mu g \cdot m^{-3}$, whereas RSP measurements in the same section of the aircraft on non-smoking flights averaged 35 to 40 $\mu g/m^3$.

(3) Hammond *et al* measured personal exposures to RSP in several hundred railroad workers.[46] Mean calculated ETS-derived exposures to RSP for railroad office workers averaged over 90 $\mu g/m^3$; by comparison, all other sources of RSP for these workers exposed to diesel exhaust averaged only 39 $\mu g/m^3$. Vaughn and Hammond measured weekly average nicotine concentrations in offices in a modern office building using both active and passive samplers.[49] Before the smoking control policy, nicotine vapour concentrations at non-smokers' desks were about 2 $\mu g/m^3$; they were reduced by 95% after a smoking ban was implemented, in good agreement with the findings of Stillman *et al*.[21]

(11) Markers for ETS in body fluids, such as nicotine and its metabolite, cotinine, are not reliable indicators of ETS exposure. Background levels of nicotine in body fluids are due to diet or persistence of nicotine in the indoor environment and there is no good way to measure uptake of ETS in the body[12]

REBUTTAL

The 1986 Surgeon General's report[14] stated that absorption of nicotine by non-smokers has been shown in several countries, suggesting that exposure to ETS is common and that the concentration of nicotine and its metabolite, cotinine, increases in body fluids with self reported ETS exposure. The 1987 IARC report on passive smoking concluded that cotinine has been shown to be a valid and sensitive marker of current exposure to ETS during daily life.[50] As to the appropriateness of nicotine and cotinine in body fluids as markers for ETS exposure, according to the IARC, cotinine in plasma, urine, or saliva is sufficiently sensitive and specific to identify passive smokers.[51] Recent studies have reported that nicotine in air from ETS is 60%, to 80% absorbed by non-smoking women.[51] Perez-Stable *et al* state that a daily consumption of several pounds of vegetables (*Solanecae* family) would be necessary to produce measurable serum cotinine levels.[52] while Tunstall-Pedoe *et al* found that serum cotinine showed little or no association with self reported daily tea consumption.[53]

(12) Non-smokers' doses of nicotine and RSP from ETS are too small, when expressed in units of cigarette equivalents, to result in disease[54]

REBUTTAL

The Surgeon General's Report considered the concept of "cigarette equivalents" as a measure of exposure and as an index of risk.[11] It concluded that the cigarette equivalent calculation, to be meaningful, would have to be done separately for each constituent. When this is done, different constituents have different equivalents. These limitations make extrapolation from atmospheric measures to cigarette equivalents of disease risk "a complex and potentially meaningless process." Moreover, nicotine and RSP are only surrogates for more than 4700 other chemical compounds, among which are 43 known carcinogens.[18,37]

(13) Smoking by a spouse is not a good surrogate for ETS exposure, flawing epidemiological studies of passive smoking and lung cancer or heart disease based on spousal smoking[11]

REBUTTAL

In an international collaborative study of exposure of 1369 non-smoking women to ETS Riboli *et al* examined the relation between smoking by a spouse and urinary cotinine levels as an indicator of exposure to ETS.[55] These investigators found that cotinine values were significantly higher for women exposed to ETS from their husband than from other sources; they also found that questionnaires in epidemiological studies based on self reports of a spouse's smoking in fact identified a most exposed population.

(14) Respirable suspended particulate matter (RSP) from ETS is not an important source of indoor air pollution

The tobacco industry has argued that source-apportionment studies show ETS–RSP to be only a small fraction of total suspended particulate matter (TSP); assertions to the contrary are not supported.[11]

REBUTTAL

Leaderer and Hammond measured weekly average vapour phase nicotine and RSP concentrations in 96 residences. Vapour phase nicotine measurements were found to be closely related to number of cigarettes smoked and highly predictive of RSP generated by tobacco combustion.[25] The mean RSP background in the absence of measurable nicotine was found to be 15·2 (7) $\mu g/m^3$. The mean RSP value in the presence of nicotine was 44·1 (30) $\mu g/m^3$. Weekly mean nicotine concentration in the 47 residences with finite nicotine values was 2·17 $\mu g/m^3$ (2·43) $\mu g/m^3$.

Miesner *et al* studied RSP and nicotine work-week average concentrations in 21 workplaces.[56] In 11 workplaces where nicotine values were above zero the average nicotine concentration was 6·59 (7·6) $\mu g/m^3$, and when the two smoking rooms were subtracted, 4·42 $\mu g/m^3$ (4·8) $\mu g/m^3$. The average RSP concentration in the smoking areas was 110 $\mu g/m^3$ (120) $\mu g/m^3$, and in the non-smoking areas was 25 $\mu g/m^3$.

Both chamber and field studies have shown that tobacco combustion has a major impact on the mass of suspended particulate matter in occupied spaces in the size range $< 2{\cdot}5$ µm, defined here as RSP. RSP is a major component of ETS.[56–60] Even under conditions of low smoking rates, easily measurable increases in RSP have been recorded above background levels. The term RSP, however, encompasses a broad range of particulates of varying chemical composition and size emanating from a number of sources (outdoors, cooking indoors, kerosene heaters, etc).[25] The apportionment of RSP indoors depends primarily on the presence of these other sources; however, there are few indoor sources generating concentrations which approach in strength those due to ETS. There seems to be little variability between brands of cigarettes or tobaccos for RSP emissions, although cigars will produce greater emissions than cigarettes.[58] Thus, it may be inferred from a comparison of smoking and non-smoking buildings that the bulk of the RSP found in buildings where there is smoking is due to ETS. For example, by combining the data of First,[59] Leaderer *et al*,[60] and Repace and Lowrey[57,58] for a total of 42 smoking buildings and 21 non-smoking buildings, the weighted average RSP level in the smoking buildings is 262 $\mu g/m^3$ compared with 36 $\mu g/m^3$ in the non-smoking buildings, suggesting that about 85% of the indoor RSP levels in those buildings is due to ETS. Most of the buildings were public access buildings. Repace and Lowrey have suggested that the average population exposure to RSP from ETS is of the order of 1·4 mg/day and that the most exposed non-smokers have 14 mg/day of exposure.[61] Although Guerin *et al* have suggested that ETS contributes only 10% to 50% of RSP in buildings,[62] this seems to be an artifact of averaging over periods when there is no smoking. Modelling and measurement in chambers and field studies suggest that the fraction contributed *during smoking* is actually between 80% and 90%.[42,45,57,58]

Recent research has shown that in large office buildings there are many pathways for floor to floor air communication, particularly return air plenums and lift shafts, where the existence of such pathways can cause a building's air exchange characteristics to closely approximate those of a single, large, open space[63]; it does not require unusual numbers or sizes of openings to create these conditions (A Persily, National Institute of Standards and Technology, personal communication, 1987). This implies that ETS may diffuse throughout a large office building, exposing non-smokers even in private offices. Nicotine measurements in office buildings support this observation.[50,64]

(15) Studies of Sterling *et al*[45] and Turner *et al*[46] show little or no difference between RSP levels in smoking and non-smoking offices[41,67]

The tobacco industry and its consultants frequently tout these papers, which conclude that "ETS does not appear to contribute significantly to a buildup of contaminants in offices or to be associated with cases of building illness"[45] and there is no significant difference between smoking and non-smoking RSP levels in offices.[45,66]

REBUTTAL

In the case of Sterling *et al*,[45] a later corrective editorial and two letters to the editor in the same journal found that the paper's conclusions[65] were not justified by the data presented and were based largely on unpublished data.[34,67,68] Insofar as the work of Turner *et al* is concerned,[66] it has been contradicted by the findings of Turk *et al*[69] and was found to be flawed in its definition of a smoking area.[70]

(16) Risks from exposures to ETS on aircraft are not significant[41,71]

The tobacco industry often cites a paper by Oldaker and Conrad (RJ Reynolds Tobacco Company) which concludes that exposures of non-smokers on aircraft are "orders of magnitude lower than smoking a single cigarette," and that simple separation of smokers from non-smokers on aircraft "significantly reduces ETS exposure of nonsmokers seated in non-smoking sections of aircraft."[71]

REBUTTAL

The significance of the risks of exposures to ETS on commercial aircraft has been investigated by Nagda *et al* for the US Department of Transportation,[47] as well as by an interdisciplinary team of investigators including researchers from the National Cancer Institute, the Environmental Protection Agency, and academia.[72] Lifetime risks from ETS exposure estimated by the Department of Transportation study for non-smoking crew members subject to 20 years of occupational exposure were 12 to 16 per 100000,[47] a level which is more than an order of magnitude higher than the maximum *de minimis* (or "acceptable") risk level used by federal agencies for environmental carcinogens in air, water, or food.[16,73] Repace estimated proportionally similar risks.[74] Mattson *et al* found that ETS exposure on aircraft produced measurable levels of cotinine in the urine of passengers and crew and that objective changes in eye and nose symptoms as well as passengers' perceptions of annoyance and smokiness were significantly related to ETS exposures.[75]

(17) Statements by public health authorities concerning health effects of ETS on children, on people with asthma, or related to heart disease and respiratory disease are not scientifically justified[7,11,12]

REBUTTAL

The technical issues related to heart disease in adults and respiratory symptoms and asthma in adults and children from passive smoking have been addressed by the Surgeon General and the National Research Council, both of which concluded, on the basis of biological plausibility and a wealth of epidemiological studies, that children exposed to ETS suffer increased rates of respiratory infections and symptoms and had slightly diminished lung function.[14,25]

As far as heart disease is concerned, in 1986, when the Surgeon General's and National Research Council's Reports were issued, there was little information available linking passive smoking to heart disease.[14,25] However, since then, a substantial body of evidence has accumulated. Glantz and Parmley reviewed the epidemiology, physiology, and biochemistry of passive smoking and heart disease.[75] They found that 11 epidemiological studies, performed in various places, reflect a 30% increase in risk of death from ischaemic heart disease or myocardial infarction in non-smokers living with smokers and that the larger studies show a dose-response effect. Based on this evidence, the American Heart Association's Council on Cardiopulmonary and Critical Care recently concluded that ETS is "a major preventable cause of cardiovascular disease and death," resulting in > 40000 deaths per year.[76] An investigator with the National Institute of Occupational Safety and Health (NIOSH) estimated that the individual lifetime excess risk of heart disease death due to ETS is one to three per 100, compared with the much lower excess risk (one death per 100000) often used in determining environmental limits for other toxins.[77]

The epidemiological studies on heart disease are complemented by physiological and biochemical data showing that ETS adversely affects platelet function and damages arterial epithelium, increasing the risk of heart disease. ETS also exerts adverse effects on exercise capability of healthy people and those with heart disease by reducing the body's ability to deliver and use oxygen. In animal experiments ETS depresses cellular respiration in mitochondria. Polycyclic aromatic hydrocarbons in ETS also might accelerate, and may initiate, the development of atherosclerotic plaque. Non-smokers seem to be more sensitive to the irritating effects of tobacco smoke than smokers, suggesting non-linear effects in dose-response.[78] A recent study by Sun *et al* has experimentally shown the induction of atherosclerotic plaques in rabbits exposed to ETS.[79] Moreover, there is at least one case where workers compensation is being paid for heart disease induced by passive smoking. A non-smoking waiter who suffered a heart attack received an $85000 settlement in a workers compensation case in California.[80]

(18) Eight scientific symposia on ETS concluded that ETS was a non-problem[11]

REBUTTAL

These eight symposia, sponsored by the tobacco industry, emphasise the self serving conclusions from papers invited by the industry, presented largely by employees and consultants of the industry, and not subjected to scientific peer review.[19] (Legitimate papers were also invited to provide a veneer of respectability.) These industry symposia are contradicted by an equal number of risk assessments of ETS exposures and lung cancer published in peer reviewed scientific papers. Repace and Lowrey reviewed nine risk assessments of passive smoking and lung cancer and found that the average among eight of the nine was 5000 (2500) lung cancer deaths per year, corresponding to about 24% of the annual lung cancer deaths among non-smokers (the 9th estimated that the risk was < 1%).[15] In a 10th risk assessment, Vainio and Partanen have similarly estimated that 20% to 30% of the lung cancer deaths in non-smokers are due to passive smoking.[81] Thus, the overwhelming majority of risk assessments of passive smoking and lung cancer published in scientific papers contradict the tobacco industry symposia and show ETS to be a major public health problem.

(19) Primary lung cancer in non-smokers is an uncommon disease; most causes are unknown but identified risk factors include radon, diet, and genetics. Most of the studies of passive smoking show a high correlation with adenocarcinoma; there is no correlation between adenocarcinoma and active smoking[42,83]

REBUTTAL

To the contrary, smoking has been related to all four major types of lung cancer: epidermoid, small cell, large cell, and adenocarcinoma; adenocarcinomas have also been induced experimentally by inhalation of dilute tobacco smoke in animals.[37,44] Although studies in different countries have produced different strengths of association between adenocarcinoma and passive smoking, one histological type does not appear to the exclusion of others. While Fontham *et al* found a stronger association between passive smoking and adenocarcinoma than for other lung cancers,[26] Kalandidi *et al*, in their study of passive smoking and lung cancer, found an increase in all histological types of cancer, but less so for adenocarcinoma.[45] Thus the histological relationship between passive smoking and adenocarcinoma does not cast doubt on a causal relationship. Although genetics undoubtedly plays some part in the induction of lung cancer,[46] the major preventable cause of lung cancer in the world today is smoking.

Diet has at best a second order effect on lung cancer.[37] Dietary influence on lung cancer has been evaluated in the context of passive smoking. In his cohort study of passive smoking in 91 540 women, Hirayama found a 12% decline in risk of lung cancer among non-smoking women who ate green-yellow vegetables daily but a 125% increase in lung cancer from passive smoking, a tenfold greater effect.[47] Kalandidi *et al* performed a case-control study of passive smoking and lung cancer, enrolling 160 women with lung cancer and 160 controls with orthopaedic conditions; high consumption of fruits was inversely related to lung cancer risk, with a relative risk of 0·27 (95% confidence interval 0·10 to 0·74), whereas marriage of a non-smoking woman to a smoker was associated with a relative risk of 2·1 (1·1 to 4·1).[45] The reported associations of lung cancer risk with passive smoking and reduced fruit intake were independent and did not confound each other.

Insofar as radon is concerned, there is no intrinsic reason for radon emissions to be higher in homes of passive smokers than in homes of unexposed non-smokers. Moreover, radon accounted for no more than about a quarter of deaths from lung cancer among non-smokers in 1990.[15]

(20) Misclassification of smokers as non-smokers can entirely account for the observed risks of passive smoking, and dosimetric estimates of risk conflict with epidemiological estimates of risk[48,49]

REBUTTAL

Lee has suggested that misclassification of smokers as non-smokers can numerically account for the observed risk of passive smoking and lung cancer[48]; however, Wells observed that Lee mixes male and female data, uses suspect sources for estimating misclassification rates, and postulates misclassification rates both in Asian and US women which are not supported by the data.[50] Moreover, the world's largest study of passive smoking and lung cancer to date, by Fontham *et al*,[26] assessed misclassification rates and found that only 0·8% of cases, compared with 2·6% of controls with colon cancer and 2·0% of population controls, were misclassified as non-smokers, indicating that the net effect of misclassification in this study was to bias the odds ratio downwards towards a null effect rather than to increase it.

Dosimetric estimates of risk conflict with epidemiological estimates of risk only if the dose-response curve from active to passive smoking is linear – it is not.[73]

(21) Tobacco smoke in the air is only a symptom of bad ventilation. According to this argument, ETS decreases very rapidly after smoking, does not persist in buildings, and, if uniformly distributed throughout a building, has negligible impact on non-smoking areas with good ventilation.[7]

REBUTTAL

The essence of this argument is that ventilation is the best control measure for ETS, which is equivalent to saying that dilution is the solution to ETS pollution. In a steady state condition, however, the concentration of tobacco smoke in buildings is directly pro-

portional to the density of smokers (number of smokers per unit building volume) and inversely proportional to the building air exchange rate. This means that tobacco smoke in the air is not simply a function of "good" or "bad" ventilation, but also a function of the number of smokers in the space and the space volume. If there are three or more smokers in a room, a steady stream of smoke will be generated; the concentration of smoke will increase until equilibrium is attained and will not change unless the smoking rate changes.

This means that tobacco smoke concentrations can be eliminated by eliminating the source, but only reduced by increasing the air exchange rate. Ventilation can reduce the risk of ETS, but if it does not reduce this risk to an acceptable level the reduction is clearly inadequate to protect non-smokers. Thus, ventilation-based controls for ETS cannot be considered as adequate control measures in the absence of health-based national indoor air quality standards for the disease states caused by passive smoking. Currently there are no national standards. We have estimated, however, that to reduce the risk of lung cancer from passive smoking to a *de minimis* or "acceptable" level by applying federal standards for regulation of environmental carcinogens would require impractical amounts of ventilation or prohibitive costs for air cleaning.[41] We also estimated that at the ASHRAE (American Society of Heating, Refrigerating, and Air-Conditioning Engineers) Ventilation Standard of 20 cubic feet per minute per occupant (cfm per occupant) (10 l/s/occupant) the lung cancer risk to non-smokers in a typical workplace would exceed the maximum *de minimis* level by more than two orders of magnitude.[10,91] In existing buildings it is doubtful whether greater increases in ventilation rates than by a factor of two above design levels are even possible without major renovation work, and control of ETS by aircleaning would be uneconomical, costing more than $30000 per smoker in 1984 dollars.[41]

ETS in typical buildings does not decrease rapidly after smoking. The mean life for removal of a pollutant from a space is defined as the time it takes for the pollutant concentration to decrease to 1/e of its initial value (where e is the base of natural logarithms). It takes three mean lives for 95% of the smoke to be removed from a building after smoking ceases, where the mean life (in hours) for removal for RSP from ETS is equal to the reciprocal of the air exchange rate (expressed in units of air changes per hour).[45] For example, at an air exchange rate of 0·25 air changes per hour (ach), typical of a very tight dwelling or commercial building, it would take 12 hours for 95% of the smoke to be removed, and at the ASHRAE standard of 20 cfm/occ (about 0·84 ach assuming a 10 ft (2·83 m) ceiling) it would take 3·6 hours for 95% of the smoke to be removed from the building.[45]

(22) The American Society of Heating, Refrigerating, and Air-Conditioning Engineers (ASHRAE) recommendation of 10 litres of outdoor air per second per occupant (20 cubic feet per minute per building occupant) is adequate ventilation to deal with "moderate" levels of smoking[7,12,41]

REBUTTAL

North American design air exchange rates are set by ASHRAE Standard 62-1989[92] and (insofar as ETS is concerned) are based on design occupancy. These rates are similar to European air exchange rates.[12] The ASHRAE standard states that "supply rates of... outdoor air required for acceptable indoor air quality ...have been chosen to control... carbon dioxide and other contaminants with an adequate margin of safety and to account for... a moderate amount of smoking." Moderate smoking is not defined in the standard. In fact, the treatment of ETS by ASHRAE Standard 62-1989 was adversely influenced by the tobacco industry.[93]

ASHRAE ventilation rates are not health-based standards designed to control tobacco smoke to acceptable levels, and compliance with the ventilation rates specified by the ASHRAE standard does not ensure avoidance of health and welfare effects in exposed non-smokers, as ASHRAE acknowledges.[92] In fact, data reported by Turk *et al*[69] show that for a group of 38 Pacific Northwest buildings whose measured air exchange rates were on average 70% higher than prescribed by ASHRAE Standard 62-1989 the RSP levels in smoking buildings averaged 40% higher than the level of EPA's health-based PM_{10} standard.[70] Furthermore, at the "moderate" smoking (two cigarettes per smoker-hour; J Janssen, ASHRAE Standard 62 Committee, personal communication) and ventilation rates specified by the ASHRAE standard, risk assessment has indicated that the attendant risk to non-smoking office workers would be 226 times the maximum acceptable level for airborne carcinogens such as ETS.[10,70,91]

(23) Separation of smokers and non-smokers on different ventilation systems or total bans on smoking are not the only control options for ETS; separate smoking and non-smoking areas are adequate to minimise exposure[7]

The tobacco industry argues that non-smoking sections in restaurants are adequate control measures. It argues that because ETS is not persistent in buildings, smokers and non-smokers may share the same space at the same time. It also argues that ventilation with directional flow yields the same results as bans and that a broader range of control options other than separate ventilation and source control is needed.

REBUTTAL

Non-smoking sections in restaurants do not eliminate non-smoking patrons' exposures to ETS.[45] Moreover, they may actually increase exposures for restaurant service workers.

Separation in time of non-smokers' and smokers' use of the same space will not be effective unless it is at least three mean-lives (3·6 hours, at ASHRAE standard ventilation (20 cfm/occupant) for an office)[15] after the end of smoking. Also, ETS absorbed on surfaces will expose non-smokers to unpleasant smells. Directional flow of ventilation from a non-smoking to a smoking area (so-called plug flow) can yield greater reductions in exposure than simple separation, but it too cannot eliminate non-smokers' exposures because of backstreaming and recirculation of ETS, and it is impractical in existing buildings without major structural and ventilation system modifications. Hammond *et al* measured nicotine and RSP in two employee smoking lounges at the University of Massachusetts.[14] RSP levels varied between 220 and 350 $\mu g/m^3$ during smoking, with associated nicotine levels from 40 to 70 $\mu g/m^3$. After charcoal filter air cleaners were installed nicotine levels were virtually unchanged, and RSP levels varied between 100 and 310 $\mu g/m^3$, an inconsequential reduction.

Conclusion

In a concerted effort to influence employers, regulators, and other policy makers to resist non-smokers' demands for smoke-free workplaces, the multinational tobacco industry has mounted a worldwide public relations campaign with scientific overtones asserting that the health effects data on ETS are inconclusive, that non-smokers are exposed to trivial amounts of tobacco smoke, and that other indoor pollutants are far more important than ETS. The tobacco industry also asserts that workplace smoking policies such as "common courtesy," providing designated smoking areas on the same ventilation system, and increased ventilation or air cleaning are adequate to control ETS in workplaces and are also preferable to smoking bans. This public relations effort is a continuation of the industry's age old strategy of denying the health consequences of tobacco use. We have shown that assertions by the tobacco industry on the risks and control of ETS lack scientific credibility. Employers and policy makers considering the formulation of effective workplace smoking policies should be cateful to use credible scientific information from reliable sources whose mission is the protection of public health.

1 Gallup Organisation. *Majority backs ban on smoking in public places. Gallup Report* No 258. Feb 1987. Gallup Organisation, Princeton, New Jersey, USA.

2 Directorate of Compliance Programs, Assistant Secretary for Occupational Safety and Health, Occupational Safety and Health Administration. *Indoor air quality*. Washington, DC. 22 Oct 1990. OSHA Notice CPL 2-2.

3 Comments submitted to the Public Docket: *Environmental tobacco smoke: a guide to the development of effective workplace smoking policies, 1990*. Washington, DC. US Environmental Protection Agency, May 1990 external review draft. (EPA 400/6-90/004.)

4 Comments submitted to the Public Docket: *Health effects of passive smoking: assessment of lung cancer in adults and respiratory disorders in children, 1991*. Washington, DC. US Environmental Protection Agency, May 1990 external review draft. (EPA 600/6-90/006A.

5 Tobacco Institute. *Cooperation is better than legislation*. Undated.

6 Davis RM, Boyd GM, Shoenborn CA. "Common courtesy" and the elimination of passive smoking: results of the 1987 National Health Interview Survey. *JAMA* 1990; 263: 2208–10. (Errata message published in *JAMA* 1990; 263: 3025.)

7 Tobacco Institute. Comments submitted to the Public Docket.[4,5]

8 National Institute for Occupational Safety and Health. *Environmental tobacco smoke in the workplace: lung cancer and other health effects*. NIOSH: Cincinnati, June 1991. (Current Intelligence Bulletin No 54.)

9 US Environmental Protection Agency. *Indoor Air Facts No 5. Environmental tobacco smoke*. Washington, DC: EPA, 1989.

10 Repace JL, Lowrey AN. An indoor air quality standard for ambient tobacco smoke based on carcinogenic risk. *NY State J Med* 1985 85: 381–3.

11 Phillips Morris, Inc. Comments submitted to the Public Docket.[4,5]

12 RJ Reynolds Tobacco Co. Comments submitted to the Public Docket.[4,5]

13 Lewtas J. *et al. Indoor air – health. Human exposure and dosimetry of environmental tobacco smoke*. Research Triangle Park, North Carolina: Health Effects Research Laboratory, Office of Research and Development, US Environmental Protection Agency, 1990. (US EPA/600/1-90/008.)

14 US Department of Health and Human Services, Public Health Service. *The health consequences of involuntary smoking. A report of the Surgeon General*. Washington, DC: DHHS, 1986. (DHHS Publication No (CDC) 87-8398.)

15 Repace JL, Lowrey AH. Risk assessment methodologies for passive-smoking-induced lung cancer. *Risk Analysis* 1990; 10: 381–3.

16 Anderson I. Judge backs case against passive smoking. *New Scientist* 1991 Feb 16.

17 Reinken J. *Thru the smoke screen*. Wellington: New Zealand Ministry of Health, 1990.

18 International Agency for Research on Cancer. IARC monograph on the evaluation of the carcinogenic risk of chemicals to humans. Tobacco smoking. Lyons: IARC, 1986: 314. (IARC monograph No 38, vol 38.)

19 Chapman S, Borland R, Hill D, Owen N, Woodward S. Why the tobacco industry fears the passive smoking issue. *Int J Health Serv* 1990; 20: 417–27.

20 Millar WJ. Evaluation of the impact of smoking restrictions in a government work setting. *Can J Public Health* 1988; 79: 379–82.

21 Stillman FA, Becker DM, Swank RT, *et al*. Ending smoking at the Johns Hopkins medical institutions: an evaluation of smoking prevalence and indoor air pollution. *JAMA* 1990; 264; 1565–9.

22 Riboli E, Preston-Martin S, Saracci R, Haley NJ, Trichopoulos D, Becher H, *et al*. Exposure of nonsmoking women to environmental tobacco smoke, a ten-country collaborative study. *Cancer Causes and Control* 1990; 1: 243–52.

23 Cummings KM, Mahoney M, Bhargava AK, McElroy PD, Marshall JR. Measurement of current exposure to environmental tobacco smoke. *Arch Environ Health* 1990; 45: 74–9.

24 Cummings KM, Markello SJ, Mahoney MC, Marshall JR. Measurement of lifetime exposure to passive smoke. *Am J Epidemiol* 1989; 130: 122–32.

25 National Research Council. *Environmental tobacco smoke – measuring exposures and assessing health effects*. Washington, DC: National Academy Press, 1986.

26 Fontham ETH, Correa P, Wu-Williams A, Reynolds P, Greenberg RS, Buffler PA, *et al*. Lung cancer in nonsmoking women: a multicenter case-control study. *Cancer Epidemiology Biomarkers and Prevention* 1991; 1: 35–43.

27 White JR, Froeb HF. Small airways dysfunction in nonsmokers chronically exposed to tobacco smoke. *N Engl J Med* 1980; 302: 720–3.

28 Masi MA, Hanley JA, Ernst P, Becklake MR. Environmental exposure to tobacco smoke and lung function in young adults. *Am Rev Respir Dis* 1984; 138: 296–9.

29 Lewis TR. Comments submitted to the Public Docket.[4]

30 US Centers for Disease Control. Discomfort from environmental tobacco smoke among employees at worksites with minimal smoking restrictions – United States, 1988. *MMWR* 1992; 41: 352–4.

31 Barad CB. Smoking on the job: the controversy heats up. *Occup Health Saf* 1979; 48: 21–31.

32 Repace JL. Risks of passive smoking. In: Gibson M, ed. *To breathe freely*. Totowa, New Jersey: Rowman and Allanheld, 1985.

33 Melius J, Wallingford K, Keenlyside R, Carpenter J. Indoor air quality: the NIOSH experience. *Am Conf Govt Ind Hyg* 1984; 10: 3–7.

34 Repace JL, Lowrey AH. Environmental tobacco smoke and indoor air quality in modern office work environments. *J Occup Med* 1987; 29: 628–9.

35 Rodriguez JF. Comments submitted to the Public Docket.[4]

36 Dube MF, Green CR. Methods of collection of smoke for analytical purposes. *Recent Advances in Tobacco Science* 1982; 8: 42–102.

37 US Department of Health and Human Services. *Reducing the health consequences of smoking: 25 years of progress*. A report of the Surgeon General, 1989. Atlanta, Georgia: Centers for Disease Control, Office on Smoking and Health, 1989. (DHHS Publication No (CDC) 89-8411.)

38 Adams J, O'Mara-Adams K, Hoffmann D. Toxic and carcinogenic agents in undiluted mainstream smoke and sidestream smoke of different types of cigarettes. *Carcinogenesis* 1987; 8: 729–31.

39 National Research Council. Health risks of radon. Washington, DC: National Academy Press. 1988. (BIER IV report.)

40 US Department of Health and Human Services. *The health consequences of smoking: cancer and chronic lung disease in the workplace. A report of the Surgeon General.* Washington, DC: Public Health Service. 1985. DHHS Publication No (PHS) 85-50207.

41 Healthy Buildings International. Comments submitted to the Public Docket.[4]

42 Repace JL. Indoor concentrations of environmental tobacco smoke: field surveys. In: O'Neill IK, Brunnemann KD, Dodet B, Hoffmann D, eds. *Environmental carcinogens: methods of analysis and exposure measurement.* Vol 9. *Passive smoking.* Lyons: International Agency for Research on Cancer, 1987: 141–62.

43 Leaderer BP. Assessing exposures to environmental tobacco smoke. *Risk Analysis* 1990; 10: 19–26.

44 Coghlin J, Hammond SK, Gann PH. Development of epidemiologic tools for measuring environmental tobacco smoke exposure. *Am J Epidemiol* 1989; 130: 696–704.

45 Repace JL. Indoor concentrations of environmental tobacco smoke: models dealing with effects of ventilation and room size. In: O'Neill IK, Brunnemann KD, Dodet B, Hoffmann D, eds. *Environmental carcinogens: methods of analysis.* Vol 9. *Passive smoking.* Lyons: International Agency for Research on Cancer, 1987: 25–40.

46 Nelson PA, Quant FR, Sem GJ. Experimental measurement of aerosol concentrations in offices. *Environment International* 1982; 8: 223–7.

47 Nagda NL, Fortmann RC, Koontz MD, Baker SR, Ginevan ME. *Airliner cabin environment: contaminant measurements health risks, and mitigation options.* US Department of Transportation Report No DOT P 15895, 1989.

48 Hammond SK, Smith TJ, Woskie SR, Leaderer BP, Bettinger N. Markers of exposure to diesel exhaust and cigarette smoke in railroad workers. *Am Ind Hyg Assoc J* 1988; 49: 516–22.

49 Vaughn WM, Hammond SK. Impact of 'designated smoking area' policy on nicotine vapor and particle concentrations in a modern office building. *J Air Waste Manage Assoc* 1990; 40: 1012–7.

50 O'Neill IK, Brunnemann KD, Dodet B, Hoffmann D, eds. *Passive smoking.* Lyons: International Agency for Research on Cancer, 1987. (IARC Scientific Publications No 81.)

51 Iwasa A, Aiba M, Kira S. Respiratory nicotine absorption in nonsmoking females during passive smoking. *Int Arch Occup Environ Health* 1991; 63: 139–43.

52 Perez-Stable EJ, Marin G, Marin BV, Benowitz NL. Misclassification of smoking status by self reported cigarette consumption. *Am Rev Respir Dis* 1992; 145: 53–7.

53 Tunstall-Pedoe H, Woodward M, Brown CA. Tea drinking, passive smoking, and serum cotinine in the Scottish heart health study. *J Clin Epidemiol* 1991; 44: 1411–4.

54 Holcomb L. Comments submitted to the Public Docket.[4]

55 Leaderer BP, Hammond SK. An evaluation of vapor-phase nicotine and respirable suspended particle mass as markers for environmental tobacco smoke. *Environmental Science and Technology* 1991; 25: 770–7.

56 Miesner EA, Rudnick SN, Preller L, Nelson W. Particulate and nicotine sampling in public facilities and offices. *JAPCA* 1989; 39: 1577–82.

57 Repace JL, Lowrey AH. Indoor air pollution, tobacco smoke, and public health. *Science* 1980; 208: 464–72.

58 Repace JL, Lowrey AH. Tobacco smoke, ventilation, and indoor air quality. *ASHRAE Transactions* 1982; 88: 894–914.

59 First MW. Environmental tobacco smoke measurement: retrospect and prospect. *Eur J Respir Dis* 1984; 5 (suppl): 9–16.

60 Leaderer BP, Cain WS, Isseroff G, Berglund LG. Ventilation requirements in buildings. II. Particulate matter and carbon monoxide from cigarette smoking. *Atmos Environ* 1984; 18: 99–106.

61 Repace JL, Lowrey AH. A quantitative estimate of nonsmokers' lung cancer risk from passive smoking. *Environment International* 1985; 11: 3–22.

62 Guerin MR, Jenkins RA, Tomkins BA. *The chemistry of environmental tobacco smoke: composition and measurement.* Boca Raton, Florida: Lewis, 1992.

63 Grot RA, Clark RE. Measured air infiltration and ventilation rates in 8 large office buildings. In: Trechsel H, Lagus P, eds. *ASTM Special Publication 904.* Philadelphia: ASTM, 1986: 151–83.

64 Williams DC, Whitaker JR, Jennings WG. Measurement of nicotine in building air as an indicator of tobacco smoke levels. *Environ Health Perspect* 1985; 60: 405–10.

65 Sterling T. Environmental tobacco smoke and indoor air quality in modern office work environments. the author replies. *J Occup Med* 1987; 29: 632–4.

66 Turner S. The measurement of environmental tobacco smoke in 585 office environments. *Environment International* 1992; 18: 19–28.

67 Sehmer JM. Tobacco smoke and air quality. *J Occup Med* 1987; 29: 637.

68 Kirkbride J. Tobacco smoke and air quality. *J Occup Med* 1987; 29: 637.

69 Turk BH, Grimsrud DT, Brown JT, Geisling-Sobotka KL, Harrison J, Prill RJ. Commercial building ventilation rates and particle concentrations. *ASHRAE Transactions* 1989; 95: 422–33.

70 Repace JL, Lowrey AH. Does ventilation really control environmental tobacco smoke in offices? *Environment International* 1992; 18: 311–25.

71 Oldaker GB, Conrad FC. Estimation of effect of environmental tobacco smoke on air quality within passenger cabins of commercial aircraft. *Environmental Science and Technology* 1987; 21: 994.

72 Mattson ME, Boyd G, Byar D, Brown C, Callahan JF, *et al.* Passive smoking on commercial airline flights. *JAMA* 1989; 261: 867–72.

73 Spengler JD, Treitman RD, Tosteson TD, Mage DT, Soczek ML. Personal exposures to respirable particulates and implications for air pollution epidemiology. *Environmental Science and Technology* 1985; 19: 700–7.

74 Repace JL. Are indoor air quality standards needed? In: Berglund B, Lindvall T, eds. *Healthy buildings '88.* Vol 4. *Conclusions and recommendations for healthier buildings.* Stockholm: Swedish Council for Building Research, 1991: 153–9.

75 Glantz SA, Parmley WW. Passive smoking and heart disease: epidemiology, physiology, and biochemistry. *Circulation* 1991; 83: 1–11.

76 Council on Cardiopulmonary and Critical Care, American Heart Association. *Circulation* 1992; 86: 1–4.

77 Steenland K. Passive smoking and the risk of heart disease. *JAMA* 1992; 267: 94–9.

78 Repace JL. Is the dose-response curve between tobacco smoke exposure and lung cancer linear from active to passive smoking? *Environment International* 1992; 18: 427–9.

79 Sun Y-P, Zhu B-Q, Sievers RE, Isenberg WM, Glantz SA, Parmley WW. Passive smoking increases experimental atherosclerosis in cholesterol-fed rabbits. Abstract to be presented at the annual meeting of the American Heart Association, 16–19 November 1992, New Orleans, Louisiana, USA.

80 Raeburn P. Waiter wins settlement in claim of heart disease from second-hand smoke. *Associated Press* 1990 Dec 14.

81 Vainio H, Partanen T. Population burden of lung cancer due to environmental tobacco smoke. *Mutat Res* 1989; 222: 137–40.

82 Adlkofer F. Comments submitted to the Public Docket.[4]

83 Leslie G. Comments submitted to the Public Docket.[4]

84 Morabia A, Wynder EL. Cigarette smoking and lung cancer types. *Cancer* 1991; 68: 2074–8.

85 Kalandidi A, Katsouyanni K, Voropoulou N, Bastas G, Saracci R, Trichopoulos D. Passive smoking and diet in the etiology of lung cancer among nonsmokers. *Cancer Causes and Control* 1990; 1: 15–21.

86 Bonney GE. Interactions of genes, environment, and lifestyle in lung cancer development. *J Natl Cancer Inst* 1990; 82: 1236–7.

87 Hirayama T. Cancer mortality in nonsmoking women with smoking husbands based on a large-scale cohort study in Japan. *Prev Med* 1984; 13: 680–90.

88 Lee PN. Comments submitted to the Public Docket.[4]

89 Rutsch M. Comments submitted to the Public Docket.[4]

90 Wells AJ. An estimate of adult mortality in the United States from passive smoking: a response. *Environment International* 1992; 18: 318–25.

91 Repace JL. Effect of ventilation on passive smoking in a model workplace. *Proceedings of an engineering foundation conference on management of atmospheres in tightly enclosed spaces, Santa Barbara, 17–21 October 1983.* Atlanta: American Society of Heating, Refrigerating, and Air-Conditioning Engineers, 1984.

92 American Society of Heating, Refrigerating, and Air-Conditioning Engineers. ASHRAE Standard 62-1989. Atlanta: ASHRAE, 1989.

93 Repace JL. Workshop comments on the dramatic changes in recommended outdoor air rates – what are the rationale, motives and consequences. In: Berglund B, Lindvall T, eds. *Healthy buildings '88.* Vol 4. *Conclusions and recommendations for healthier buildings.* Stockholm: Swedish Council for Building Research, 1991: 132–3.

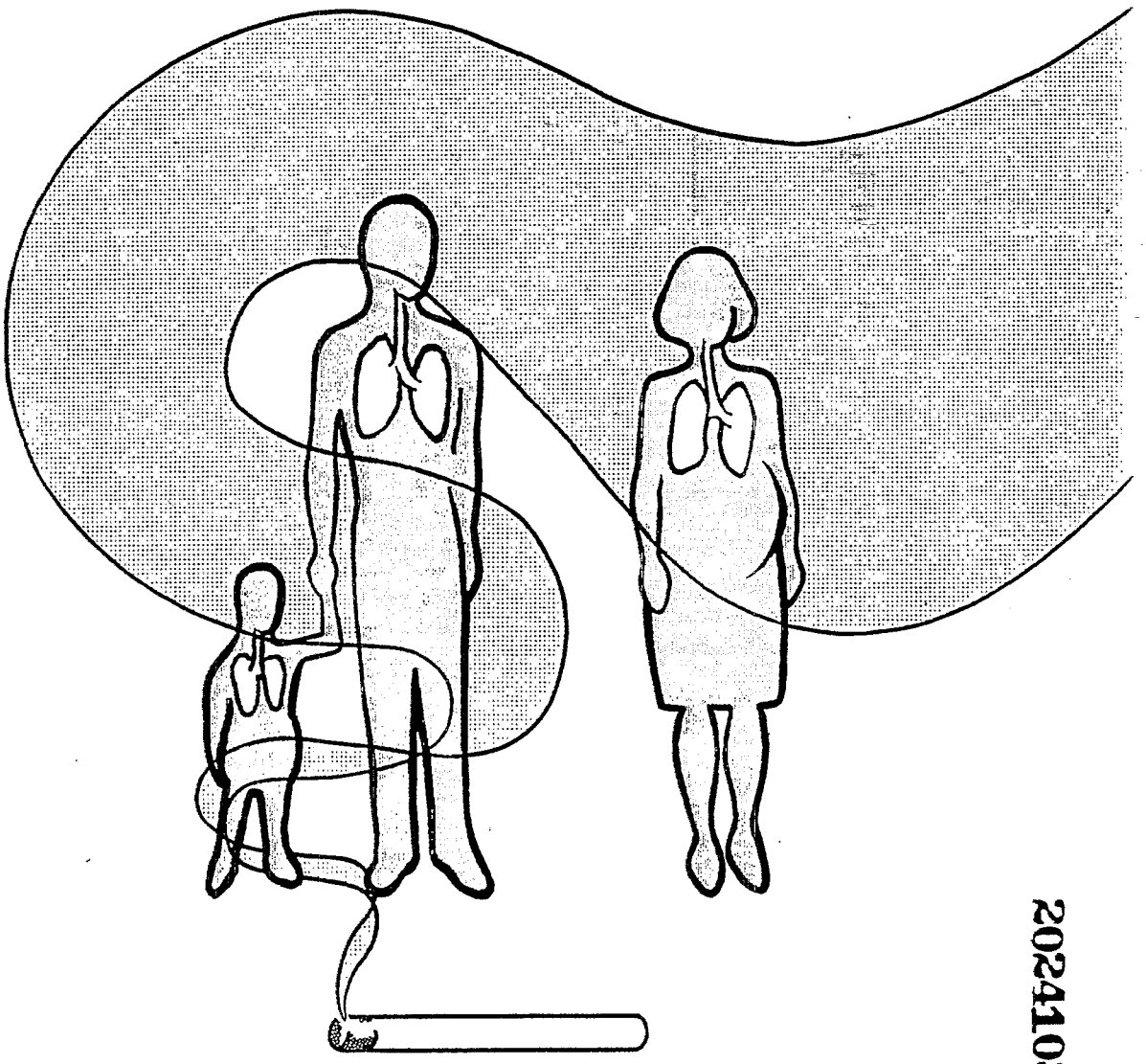
94 Hammond SK, Leaderer BP, Roche A. Collection and analysis of nicotine as a marker for environmental tobacco smoke. *Atmos Environ* 1987; 21: 457–62.

United States Environmental Protection Agency
Office of Research and Development Washington, DC 20460
Office of Air and Radiation Washington, DC 20460
EPA/600/6-90/006F
December 1992

EPA

Respiratory Health Effects of Passive Smoking:

Lung Cancer and Other Disorders



1. SUMMARY AND CONCLUSIONS

1.1. MAJOR CONCLUSIONS

Based on the weight of the available scientific evidence, the U.S. Environmental Protection Agency (EPA) has concluded that the widespread exposure to environmental tobacco smoke (ETS) in the United States presents a serious and substantial public health impact.

In adults:

- ETS is a human lung carcinogen, responsible for approximately 3,000 lung cancer deaths annually in U.S. nonsmokers.

In children:

- ETS exposure is causally associated with an increased risk of lower respiratory tract infections (LRIs) such as bronchitis and pneumonia. This report estimates that 150,000 to 300,000 cases annually in infants and young children up to 18 months of age are attributable to ETS.

- ETS exposure is causally associated with increased prevalence of fluid in the middle ear, symptoms of upper respiratory tract irritation, and a small but significant reduction in lung function.

- ETS exposure is causally associated with additional episodes and increased severity of symptoms in children with asthma. This report estimates that 200,000 to 1,000,000 asthmatic children have their condition worsened by exposure to ETS.

- ETS exposure is a risk factor for new cases of asthma in children who have not previously displayed symptoms.

1.2. BACKGROUND

Tobacco smoking has long been recognized (e.g., U.S. Department of Health, Education, and Welfare [U.S. DHEW], 1964) as a major cause of mortality and morbidity, responsible for an estimated 434,000 deaths per year in the United States (Centers for Disease Control [CDC], 1991a). Tobacco use is known to cause cancer at various sites, in particular the lung (U.S. Department of Health and Human Services [U.S. DHHS], 1982; International Agency for Research on Cancer [IARC], 1986). Smoking can also cause respiratory diseases (U.S. DHHS, 1984, 1989) and is a major risk factor for heart disease (U.S. DHHS, 1983). In recent years, there has been concern that nonsmokers may also be at risk for some of these health effects as a result of their exposure ("passive smoking") to the tobacco smoke that occurs in various environments occupied by smokers. Although this ETS is dilute compared with the mainstream smoke (MS) inhaled by active smokers, it is chemically similar, containing many of the same carcinogenic and toxic agents.

In 1986, the National Research Council (NRC) and the Surgeon General of the U.S. Public Health Service independently assessed the health effects of exposure to ETS (NRC, 1986; U.S. DHHS, 1986). Both of the 1986 reports conclude that ETS can cause lung cancer in adult nonsmokers and that children of parents who smoke have increased frequency of respiratory symptoms and acute lower respiratory tract infections, as well as evidence of reduced lung function.

More recent epidemiologic studies of the potential associations between ETS and lung cancer in nonsmoking adults and between ETS and noncancer respiratory effects more than double the size of the database available for analysis from that of the 1986 reports. This EPA report critically reviews the current database on the respiratory health effects of passive smoking; these data are utilized to develop a hazard identification for ETS and to make quantitative estimates of the public health impacts of ETS for lung cancer and various other respiratory diseases.

The weight-of-evidence analysis for the lung cancer hazard identification is developed in accordance with U.S. EPA's *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 1986a) and established principles for evaluating epidemiologic studies. The analysis considers animal bioassays and genotoxicity studies, as well as biological measurements of human uptake of tobacco smoke components and epidemiologic data on active and passive smoking. The availability of abundant and consistent human data, especially human data at actual environmental levels of exposure to the specific agent (mixture) of concern, allows a hazard identification to be made with a high degree of certainty. The conclusive evidence of the dose-related lung carcinogenicity of

MS in active smokers (Chapter 4), coupled with information on the chemical similarities of MS and ETS and evidence of ETS uptake in nonsmokers (Chapter 3), is sufficient by itself to establish ETS as a known human lung carcinogen, or "Group A" carcinogen under U.S. EPA's carcinogen classification system. In addition, this document concludes that the overall results of 30 epidemiologic studies on lung cancer and passive smoking (Chapter 5), using spousal smoking as a surrogate of ETS exposure for female never-smokers, similarly justify a Group A classification.

The weight-of-evidence analyses for the noncancer respiratory effects are based primarily on a review of epidemiologic studies (Chapter 7). Most of the endpoints examined are respiratory disorders in children, where parental smoking is used as a surrogate of ETS exposure. For the noncancer respiratory effects in nonsmoking adults, most studies used spousal smoking as an exposure surrogate. A causal association was concluded to exist for a number of respiratory disorders where there was sufficient consistent evidence for a biologically plausible association with ETS that could not be explained by bias, confounding, or chance. The fact that the database consists of human evidence from actual environmental exposure levels gives a high degree of confidence in this conclusion. Where there was suggestive but inconclusive evidence of causality, as was the case for asthma induction in children, ETS was concluded to be a risk factor for that endpoint. Where data were inconsistent or inadequate for evaluation of an association, as for acute upper respiratory tract infections and acute middle ear infections in children, no conclusions were drawn.

This report also has attempted to provide estimates of the extent of the public health impact, where appropriate, in terms of numbers of ETS-attributable cases in nonsmoking subpopulations. Unlike for qualitative hazard identification assessments, where information from many sources adds to the confidence in a weight-of-evidence conclusion, for quantitative risk assessments, the usefulness of studies usually depends on how closely the study population resembles nonsmoking segments of the general population. For lung cancer estimates among U.S. nonsmokers, the substantial epidemiology database of ETS and lung cancer among U.S. female never-smokers was considered to provide the most appropriate information. From these U.S. epidemiology studies, a pooled relative risk estimate was calculated and used in the derivation of the population risk estimates. The large number of studies available, the generally consistent results, and the condition of actual environmental levels of exposure increase the confidence in these estimates. Even under these circumstances, however, uncertainties remain, such as in the use of questionnaires and current biomarker measurements to estimate past exposure, assumptions of exposure-response linearity, and extrapolation to male never-smokers and to ex-smokers. Still, given the strength of the evidence for the lung carcinogenicity of tobacco smoke and the extensive human database from actual environmental exposure levels, fewer assumptions are necessary than

is usual in EPA quantitative risk assessments, and confidence in these estimates is rated medium to high.

Population estimates of ETS health impacts are also made for certain noncancer respiratory endpoints in children, specifically lower respiratory tract infections (i.e., pneumonia, bronchitis, and bronchiolitis) and episodes and severity of attacks of asthma. Estimates of ETS-attributable cases of LRI in infants and young children are thought to have a high degree of confidence because of the consistent study findings and the appropriateness of parental smoking as a surrogate measure of exposure in very young children. Estimates of the number of asthmatic children whose condition is aggravated by exposure to ETS are less certain than those for LRIs because of different measures of outcome in various studies and because of increased extraparental exposure to ETS in older children. Estimates of the number of new cases of asthma in previously asymptomatic children also have less confidence because at this time the weight of evidence for asthma induction, while suggestive of a causal association, is not conclusive.

Most of the ETS population impact estimates are presented in terms of ranges, which are thought to reflect reasonable assumptions about the estimates of parameters and variables required for the extrapolation models. The validity of the ranges is also dependent on the appropriateness of the extrapolation models themselves.

While this report focuses only on the respiratory health effects of passive smoking, there also may be other health effects of concern. Recent analyses of more than a dozen epidemiology and toxicology studies (e.g., Steenland, 1992; National Institute for Occupational Safety and Health [NIOSH], 1991) suggest that ETS exposure may be a risk factor for cardiovascular disease. In addition, a few studies in the literature link ETS exposure to cancers of other sites; at this time, that database appears inadequate for any conclusion. This report does not develop an analysis of either the nonrespiratory cancer or the heart disease data and takes no position on whether ETS is a risk factor for these diseases. If it is, the total public health impact from ETS will be greater than that discussed here.

1.3. PRIMARY FINDINGS

A. Lung Cancer in Nonsmoking Adults

1. Passive smoking is causally associated with lung cancer in adults, and ETS, by the total weight of evidence, belongs in the category of compounds classified by EPA as Group A (known human) carcinogens.
2. Approximately 3,000 lung cancer deaths per year among nonsmokers (never-smokers and former smokers) of both sexes are estimated to be attributable to ETS in the United States. While there are statistical and modeling uncertainties

in this estimate, and the true number may be higher or lower, the assumptions used in this analysis would tend to underestimate the actual population risk. The overall confidence in this estimate is medium to high.

B. Noncancer Respiratory Diseases and Disorders

1. Exposure of children to ETS from parental smoking is causally associated with:
   a. increased prevalence of respiratory symptoms of irritation (cough, sputum, and wheeze),
   b. increased prevalence of middle ear effusion (a sign of middle ear disease), and
   c. a small but statistically significant reduction in lung function as tested by objective measures of lung capacity.
2. ETS exposure of young children and particularly infants from parental (and especially mother's) smoking is causally associated with an increased risk of LRIs (pneumonia, bronchitis, and bronchiolitis). This report estimates that exposure to ETS contributes 150,000 to 300,000 LRIs annually in infants and children less than 18 months of age, resulting in 7,500 to 15,000 hospitalizations. The confidence in the estimates of LRIs is high. Increased risks for LRIs continue, but are lower in magnitude, for children until about age 3; however, no estimates are derived for children over 18 months.
3. a. Exposure to ETS is causally associated with additional episodes and increased severity of asthma in children who already have the disease. This report estimates that ETS exposure exacerbates symptoms in approximately 20% of this country's 2 million to 5 million asthmatic children and is a major aggravating factor in approximately 10%.
   b. In addition, the epidemiologic evidence is suggestive but not conclusive that ETS exposure increases the number of new cases of asthma in children who have not previously exhibited symptoms. Based on this evidence and the known ETS effects on both the immune system and lungs (e.g., atopy and airway hyperresponsiveness), this report concludes that ETS is a risk factor for the induction of asthma in previously asymptomatic children. Data suggest that relatively high levels of exposure are required to induce new cases of asthma in children. This report calculates that previously asymptomatic children exposed to ETS from mothers who smoke at least 10 cigarettes per day will exhibit an estimated 8,000 to 26,000 new cases of

asthma annually. The confidence in this range is medium and is dependent on the conclusion that ETS is a risk factor for asthma induction.

4. Passive smoking has subtle but significant effects on the respiratory health of nonsmoking adults, including coughing, phlegm production, chest discomfort, and reduced lung function.

This report also has reviewed data on the relationship of maternal smoking and sudden infant death syndrome (SIDS), which is thought to involve some unknown respiratory pathogenesis. The report concludes that while there is strong evidence that infants whose mothers smoke are at an increased risk of dying from SIDS, available studies do not allow us to differentiate whether and to what extent this increase is related to in utero versus postnatal exposure to tobacco smoke products. Consequently, this report is unable to assert whether or not ETS exposure by itself is a risk factor for SIDS independent of smoking during pregnancy.

Regarding an association of parental smoking with either upper respiratory tract infections (colds and sore throats) or acute middle ear infections in children, this report finds the evidence inconclusive.

1.3.1. ETS and Lung Cancer

1.3.1.1. *Hazard Identification*

The Surgeon General (U.S. DHHS, 1989) estimated that smoking was responsible for more than one of every six deaths in the United States and that it accounted for about 90% of the lung cancer deaths in males and about 80% in females in 1985. Smokers, however, are not the only ones exposed to tobacco smoke. The sidestream smoke (SS) emitted from a smoldering cigarette between puffs (the main component of ETS) has been documented to contain virtually all of the same carcinogenic compounds (known and suspected human and animal carcinogens) that have been identified in the mainstream smoke (MS) inhaled by smokers (Chapter 3). Exposure concentrations of these carcinogens to passive smokers are variable but much lower than for active smokers. An excess cancer risk from passive smoking, however, is biologically plausible.

Based on the firmly established causal association of lung cancer with active smoking with a dose-response relationship down to low doses (Chapter 4), passive smoking is considered likely to affect the lung similarly. The widespread presence of ETS in both home and workplace and its absorption by nonsmokers in the general population have been well documented by air sampling and by body measurement of biomarkers such as nicotine and cotinine (Chapter 3). This raises the question of whether any direct evidence exists for the relationship between ETS exposure and lung cancer in the general population and what its implications may be for public health. This

report addresses that question by reviewing and analyzing the evidence from 30 epidemiologic studies of effects from normally occurring environmental levels of ETS (Chapter 5). Because there is widespread exposure and it is difficult to construct a truly unexposed subgroup of the general population, these studies attempt to compare individuals with higher ETS exposure to those with lower exposures. Typically, female never-smokers who are married to a smoker are compared with female never-smokers who are married to a nonsmoker. Some studies also consider ETS exposure of other subjects (i.e., male never-smokers and long-term former smokers of either sex) and from other sources (e.g., workplace and home exposure during childhood), but these studies are fewer and represent fewer cases, and they are generally excluded from the analysis presented here. Use of the female never-smoker studies provides the largest, most homogeneous database for analysis to determine whether an ETS effect on lung cancer is present. This report assumes that the results for female never-smokers are generalizable to all nonsmokers.

Given that ETS exposures are at actual environmental levels and that the comparison groups are both exposed to appreciable background (i.e., nonspousal) ETS, any excess risk for lung cancer from exposure to spousal smoke would be expected to be small. Furthermore, the risk of lung cancer is relatively low in nonsmokers, and most studies have a small sample size, resulting in a very low statistical power (probability of detecting a real effect if it exists). Besides small sample size and low incremental exposures, other problems inherent in several of the studies may also limit their ability to detect a possible effect. Therefore, this report examines the data in several different ways. After downward adjustment of the relative risks for smoker misclassification bias, the studies are individually assessed for strength of association, both for the overall data and for the highest exposure group when exposure-level data are available, and for exposure-response trend. Then the study results are pooled by country using statistical techniques for combining data, including both positive and nonpositive results, to increase the ability to determine whether or not there is an association between ETS and lung cancer. Finally, in addition to the previous statistical analyses that weight the studies only by size, regardless of design and conduct, the studies are qualitatively evaluated for potential confounding, bias, and likely utility to provide information about any lung carcinogenicity of ETS. Based on these qualitative considerations, the studies are categorized into one of four tiers and then statistically analyzed successively by tier.

Results from all of the analyses described above strongly support a causal association between lung cancer ETS exposure. The overall proportion (9/30) of individual studies found to show an association between lung cancer and spousal ETS exposure at all levels combined is unlikely to occur by chance ($p < 10^{-4}$). When the analysis focuses on higher levels of spousal exposure, every one of the 17 studies with exposure-level data shows increased risk in the highest

exposure group; 9 of these are significant at the $p < 0.05$ level, despite most having low power, another result highly unlikely to occur by chance ($p < 10^{-7}$). Similarly, the proportion (10/14; $p < 10^{-9}$) showing a statistically significant exposure-response trend is highly supportive of a causal association.

Combined results by country showed statistically significant associations for Greece (2 studies), Hong Kong (4 studies), Japan (5 studies), and the United States (11 studies), and in that order of strength of relative risk. Pooled results of the four Western European studies (three countries) actually showed a slightly stronger association than that of the United States, but it was not statistically significant, probably due to the smaller sample size. The combined results of the Chinese studies do not show an association between ETS and lung cancer; however, two of the four Chinese studies were designed mainly to determine the lung cancer effects of high levels of other indoor air pollutants indigenous to those areas, which would obscure a smaller ETS effect. These two Chinese studies do, however, provide very strong evidence on the lung carcinogenicity of these other indoor air pollutants, which contain many of the same components as ETS. When results are combined only for the other two Chinese studies, they demonstrate a statistically significant association for ETS and lung cancer.

The heterogeneity of observed relative risk estimates among countries could result from several factors. For example, the observed differences may reflect true differences in lung cancer rates for never-smokers, in ETS exposure levels from nonspousal sources, or in related lifestyle characteristics in different countries. For the time period in which ETS exposure was of interest for these studies, spousal smoking is considered to be a better surrogate for ETS exposure in more "traditional" societies, such as Japan and Greece, than in the United States. In the United States, other sources of ETS exposure (e.g., work and public places) are generally higher, which obscures the effects of spousal smoking and may explain the lower relative risks observed in the United States. Nevertheless, despite observed differences between countries, all showed evidence of increased risk.

Based on these analyses and following the U.S. EPA's *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 1986a), EPA concludes that environmental tobacco smoke is a Group A (known human) carcinogen. This conclusion is based on a total weight of evidence, principally:

- Biological plausibility. ETS is taken up by the lungs, and components are distributed throughout the body. The presence of the same carcinogens in ETS and MS, along with the established causal relationship between lung cancer and active smoking with the dose-response relationships exhibited down to low doses, establishes the plausibility that ETS is also a lung carcinogen.

- Supporting evidence from animal bioassays and genotoxicity experiments. The carcinogenicity of tobacco smoke has been demonstrated in lifetime inhalation studies in the hamster, intrapulmonary implantations in the rat, and skin painting in the mouse. There are no lifetime animal inhalation studies of ETS; however, the carcinogenicity of SS condensates has been shown in intrapulmonary implantations and skin painting experiments. Positive results of genotoxicity testing for both MS and ETS provide corroborative evidence for their carcinogenic potential.
- Consistency of response. All 4 of the cohort studies and 20 of the 26 case-control studies observed a higher risk of lung cancer among the female never-smokers classified as ever exposed to any level of spousal ETS. Furthermore, every one of the 17 studies with response categorized by exposure level demonstrated increased risk for the highest exposure group. When assessment was restricted to the 19 studies judged to be of higher utility based on study design, execution, and analysis (Appendix A), 17 observed higher risks, and 6 of these increases were statistically significant, despite most having low statistical power. Evaluation of the total study evidence from several perspectives leads to the conclusion that the observed association between ETS exposure and increased lung cancer occurrence is not attributable to chance.
- Broad-based evidence. These 30 studies provide data from 8 different countries, employ a wide variety of study designs and protocols, and are conducted by many different research teams. Results from all countries, with the possible exception of two areas of China where high levels of other indoor air lung carcinogens were present, show small to modest increases in lung cancer associated with spousal ETS exposure. No alternative explanatory variables for the observed association between ETS and lung cancer have been indicated that would be broadly applicable across studies.
- Upward trend in exposure-response. Both the largest of the cohort studies--the Japanese study of Hirayama with 200 lung cancer cases--and the largest of the case-control studies--the U.S. study by Fontham and associates (1991) with 420 lung cancer cases and two sets of controls--demonstrate a strong exposure-related statistical association between passive smoking and lung cancer. This upward trend is well supported by the preponderance of epidemiology studies. Of the 14 studies that provide sufficient data for a trend test by exposure level, 10 were statistically significant despite most having low statistical power.
- Detectable association at environmental exposure levels. Within the population of married women who are lifelong nonsmokers, the excess lung cancer risk from

exposure to their smoking husbands' ETS is large enough to be observed, even for all levels of their spousal exposure combined. Carcinogenic responses are usually detectable only in high-exposure circumstances, such as occupational settings, or in experimental animals receiving very high doses. In addition, effects are harder to observe when there is substantial background exposure in the comparison groups, as is the case here.

- Effects remain after adjustment for potential upward bias. Current and ex-smokers may be misreported as never-smokers, thus inflating the apparent cancer risk for ETS exposure. The evidence remains statistically significant and conclusive, however, after adjustments for smoker misclassification. For the United States, the summary estimate of relative risk from nine case-control plus two cohort studies is 1.19 (90% confidence interval [C.I.] = 1.04, 1.35; $p < 0.05$) after adjustment for smoker misclassification. For Greece, 2.00 (1.42, 2.83), Hong Kong, 1.61 (1.25, 2.06), and Japan, 1.44 (1.13, 1.85), the estimated relative risks are higher than those of the United States and more highly significant after adjusting for the potential bias.
- Strong associations for highest exposure groups. Examining the groups with the highest exposure levels increases the ability to detect an effect, if it exists. Nine of the sixteen studies worldwide for which there are sufficient exposure-level data are statistically significant for the highest exposure group, despite most having low statistical power. The overall pooled estimate of 1.81 for the highest exposure groups is highly statistically significant (90% C.I. = 1.60, 2.05; $p < 10^{-6}$). For the United States, the overall pooled estimate of 1.38 (seven studies, corrected for smoker misclassification bias) is also highly statistically significant (90% C.I. = 1.13, 1.70; $p = 0.005$).
- Confounding cannot explain the association. The broad-based evidence for an association found by independent investigators across several countries, as well as the positive exposure-response trends observed in most of the studies that analyzed for them, make any single confounder highly unlikely as an explanation for the results. In addition, this report examined potential confounding factors (history of lung disease, home heat sources, diet, occupation) and concluded that none of these factors could account for the observed association between lung cancer and ETS.

1.3.1.2. *Estimation of Population Risk*

The individual risk of lung cancer from exposure to ETS does not have to be very large to translate into a significant health hazard to the U.S. population because of the large number of smokers and the widespread presence of ETS. Current smokers comprise approximately 26% of the U.S. adult population and consume more than one-half trillion cigarettes annually (1.5 packs per day, on average), causing nearly universal exposure to at least some ETS. As a biomarker of tobacco smoke uptake, cotinine, a metabolite of the tobacco-specific compound nicotine, is detectable in the blood, saliva, and urine of persons recently exposed to tobacco smoke. Cotinine has typically been detected in 50% to 75% of reported nonsmokers tested (50% equates to 63 million U.S. nonsmokers age 18 or older).

The best estimate of approximately 3,000 lung cancer deaths per year in U.S. nonsmokers age 35 and over attributable to ETS (Chapter 6) is based on data pooled from all 11 U.S. epidemiologic studies of never-smoking women married to smoking spouses. Use of U.S. studies should increase the confidence in these estimates. Some mathematical modeling is required to adjust for expected bias from misclassification of smoking status and to account for ETS exposure from sources other than spousal smoking. The overall relative risk estimate of 1.19 for the United States, already adjusted for smoker misclassification bias, becomes 1.59 after adjusting for background ETS sources (1.34 for nonspousal exposures only). Assumptions are also needed to relate responses in female never-smokers to those in male never-smokers and ex-smokers of both sexes, and to estimate the proportion of the nonsmoking population exposed to various levels of ETS. Overall, however, the assumptions necessary for estimating risk add far less uncertainty than other EPA quantitative assessments. This is because the extrapolation for ETS is based on a large database of human studies, all at levels actually expected to be encountered by much of the U.S. population.

The components of the 3,000 lung cancer deaths figure include approximately 1,500 female never-smokers, 500 male never-smokers, and 1,000 former smokers of both sexes. More females are estimated to be affected because there are more female than male nonsmokers. These component estimates have varying degrees of confidence; the estimate of 1,500 deaths for female never-smokers has the highest confidence because of the extensive database. The estimate of 500 for male never-smokers is less certain because it is based on the female never-smoker response and is thought to be low because males are generally subject to higher background ETS exposures than females. Adjustment for this higher background exposure would lead to higher risk estimates. The estimate of 1,000 lung cancer deaths for former smokers of both sexes is

considered to have the lowest confidence, and the assumptions used are thought to make this estimate low as well.

Workplace ETS levels are generally comparable with home ETS levels, and studies using body cotinine measures as biomarkers demonstrate that nonspousal exposures to ETS are often greater than exposure from spousal smoking. Thus, this report presents an alternative breakdown of the estimated 3,000 ETS-attributable lung cancer deaths between spousal and nonspousal exposures. By extension of the results from spousal smoking studies, coupled with biological measurements of exposure, more lung cancer deaths are estimated to be attributable to ETS from combined nonspousal exposures--2,200 of both sexes--than from spousal exposure--800 of both sexes. This spouse-versus-other-sources partitioning depends on current exposure estimates that may or may not be applicable to the exposure period of interest. Thus, this breakdown contains this element of uncertainty in addition to those discussed above with respect to the previous breakdown.

An alternative analysis, based on the large Fontham et al. (1991) study, which is the only study that provides biomarker estimates of both relative risk and ETS exposure, yields population risk point estimates of 2,700 and 3,600. These population risk estimates are highly consistent with the estimate of 3,000 based on the combined U.S. studies.

While there is statistical variance around all of the parameters used in the quantitative assessment, the two largest areas of uncertainty are probably associated with the relative risk estimate for spousal ETS exposure and the parameter estimate for the background ETS exposure adjustment. A sensitivity analysis that independently varies these two estimates yields population risk estimates as low as 400 and as high as 7,000. These extremes, however, are considered unlikely; the more probable range is narrower, and the generally conservative assumptions employed suggest that the actual population risk number may be greater than 3,000. Overall, considering the multitude, consistency, and quality of all these studies, the weight-of-evidence conclusion that ETS is a known human lung carcinogen, and the limited amount of extrapolation necessary, the confidence in the estimate of approximately 3,000 lung cancer deaths is medium to high.

1.3.2. ETS and Noncancer Respiratory Disorders

Exposure to ETS from parental smoking has been previously linked with increased respiratory disorders in children, particularly in infants. Several studies have confirmed the exposure and uptake of ETS in children by assaying saliva, serum, or urine for cotinine. These cotinine concentrations were highly correlated with smoking (especially by the mother) in the child's presence. Nine to twelve million American children under 5 years of age, or one-half to

two-thirds of all children in this age group, may be exposed to cigarette smoke in the home (American Academy of Pediatrics, 1986; Overpeck and Moss, 1991).

With regard to the noncancer respiratory effects of passive smoking, this report focuses on epidemiologic evidence appearing since the two major reports of 1986 (NRC and U.S. DHHS) that bears on the potential association of parental smoking with detrimental respiratory effects in their children. These effects include symptoms of respiratory irritation (cough, sputum production, or wheeze); acute diseases of the lower respiratory tract (pneumonia, bronchitis, and bronchiolitis); acute middle ear infections and indications of chronic middle ear infections (predominantly middle ear effusion); reduced lung function (from forced expiratory volume and flow-rate measurements); incidence and prevalence of asthma and exacerbation of symptoms in asthmatics; and acute upper respiratory tract infections (colds and sore throats). The more than 50 recently published studies reviewed here essentially corroborate the previous conclusions of the 1986 reports of the NRC and Surgeon General regarding respiratory symptoms, respiratory illnesses, and pulmonary function, and they strengthen support for those conclusions by the additional weight of evidence (Chapter 7). For example, new data on middle ear effusion strengthen previous evidence to warrant the stronger conclusion in this report of a causal association with parental smoking. Furthermore, recent studies establish associations between parental smoking and increased incidence of childhood asthma. Additional research also supports the hypotheses that in utero exposure to mother's smoke and postnatal exposure to ETS alter lung function and structure, increase bronchial responsiveness, and enhance the process of allergic sensitization, changes that are known to predispose children to early respiratory illness. Early respiratory illness can lead to long-term pulmonary effects (reduced lung function and increased risk of chronic obstructive lung disease).

This report also summarizes the evidence for an association between parental smoking and SIDS, which was not addressed in the 1986 reports of the NRC or Surgeon General. SIDS is the most common cause of death in infants ages 1 month to 1 year. The cause (or causes) of SIDS is unknown; however, it is widely believed that some form of respiratory pathogenesis is generally involved. The current evidence strongly suggests that infants whose mothers smoke are at an increased risk of dying of SIDS, independent of other known risk factors for SIDS, including low birthweight and low gestational age, which are specifically associated with active smoking during pregnancy. However, available studies do not allow this report to conclude whether that increased risk is related to in utero versus postnatal exposure to tobacco smoke products, or to both.

The 1986 reports of the NRC and Surgeon General conclude that both the prevalence of respiratory symptoms of irritation and the incidence of lower respiratory tract infections are higher in children of smoking parents. In the 18 studies of respiratory symptoms subsequent to

the 2 reports, increased symptoms (cough, phlegm production, and wheezing) were observed in a range of ages from birth to midteens, particularly in infants and preschool children. In addition to the studies on symptoms of respiratory irritation, 10 new studies have addressed the topic of parental smoking and acute lower respiratory tract illness in children, and 9 have reported statistically significant associations. The cumulative evidence is conclusive that parental smoking, especially the mother's, causes an increased incidence of respiratory illnesses from birth up to the first 18 months to 3 years of life, particularly for bronchitis, bronchiolitis, and pneumonia. Overall, the evidence confirms and strengthens the previous conclusions of the NRC and Surgeon General.

Recent studies also solidify the evidence for the conclusion of a causal association between parental smoking and increased middle ear effusion in young children. Middle ear effusion is the most common reason for hospitalization of young children for an operation.

At the time of the Surgeon General's report on passive smoking (U.S. DHHS, 1986), data were sufficient to conclude only that maternal smoking may influence the severity of asthma in children. The recent studies reviewed here strengthen and confirm these exacerbation effects. The new evidence is also conclusive that ETS exposure increases the number of episodes of asthma in children who already have the disease. In addition, the evidence is suggestive that ETS exposure increases the number of new cases of asthma in children who have not previously exhibited symptoms, although the results are statistically significant only with children whose mothers smoke 10 or more cigarettes per day. While the evidence for new cases of asthma itself is not conclusive of a causal association, the consistently strong association of ETS both with increased frequency and severity of the asthmatic symptoms and with the established ETS effects on the immune system and airway hyperresponsiveness lead to the conclusion that ETS is a risk factor for induction of asthma in previously asymptomatic children.

Regarding the effects of passive smoking on lung function in children, the 1986 NRC and Surgeon General reports both conclude that children of parents who smoke have small decreases in tests of pulmonary output function of both the larger and smaller air passages when compared with the children of nonsmokers. As noted in the NRC report, if ETS exposure is the cause of the observed decrease in lung function, the effect could be due to the direct action of agents in ETS or an indirect consequence of increased occurrence of acute respiratory illness related to ETS.

Results from eight studies on ETS and lung function in children that have appeared since those reports add some additional confirmatory evidence suggesting a causal rather than an indirect relationship. For the population as a whole, the reductions are small relative to the interindividual variability of each lung function parameter. However, groups of particularly susceptible or heavily exposed children have shown larger decrements. The studies reviewed

suggest that a continuum of exposures to tobacco products starting in fetal life may contribute to the decrements in lung function found in older children. Exposure to tobacco smoke products inhaled by the mother during pregnancy may contribute significantly to these changes, but there is strong evidence indicating that postnatal exposure to ETS is an important part of the causal pathway.

With respect to lung function effects in adults exposed to ETS, the 1986 NRC and Surgeon General reports found the data at that time inconclusive, due to high interindividual variability and the existence of a large number of other risk factors, but compatible with subtle deficits in lung function. Recent studies confirm the association of passive smoking with small reductions in lung function. Furthermore, new evidence also has emerged suggesting a subtle association between exposure to ETS and increased respiratory symptoms in adults.

Some evidence suggests that the incidence of acute upper respiratory tract illnesses and acute middle ear infections may be more common in children exposed to ETS. However, several studies failed to find any effect. In addition, the possible role of confounding factors, the lack of studies showing clear dose-response relationships, and the absence of a plausible biological mechanism preclude more definitive conclusions.

In reviewing the available evidence indicating an association (or lack thereof) between ETS exposure and the different noncancer respiratory disorders analyzed in this report, the possible role of several potential confounding factors was considered. These include other indoor air pollutants; socioeconomic status; effect of parental symptoms; and characteristics of the exposed child, such as low birthweight or active smoking. No single or combined confounding factors can explain the observed respiratory effects of passive smoking in children.

For diseases for which ETS has been either causally associated (LRIs) or indicated as a risk factor (asthma cases in previously asymptomatic children), estimates of population-attributable risk can be calculated. A population risk assessment (Chapter 8) provides a probable range of estimates that 8,000 to 26,000 cases of childhood asthma per year are attributable to ETS exposure from mothers who smoke 10 or more cigarettes per day. The confidence in this range of estimates is medium and is dependent on the suggestive evidence of the database. While the data show an effect only for children of these heavily smoking mothers, additional cases due to lesser ETS exposure also are a possibility. If the effect of this lesser exposure is considered, the range of estimates of new cases presented above increases to 13,000 to 60,000. Furthermore, this report estimates that the additional public health impact of ETS on asthmatic children includes more than 200,000 children whose symptoms are significantly aggravated and as many as 1,000,000 children who are affected to some degree.

This report estimates that ETS exposure contributes 150,000 to 300,000 cases annually of lower respiratory tract illness in infants and children younger than 18 months of age and that 7,500 to 15,000 of these will require hospitalization. The strong evidence linking ETS exposure to increased incidence of bronchitis, bronchiolitis, and pneumonia in young children gives these estimates a high degree of confidence. There is also evidence suggesting a smaller ETS effect on children between the ages of 18 months and 3 years, but no additional estimates have been computed for this age group. Whether or not these illnesses result in death has not been addressed here.

In the United States, more than 5,000 infants die of SIDS annually. It is the major cause of death in infants between the ages of 1 month and 1 year, and the linkage with maternal smoking is well established. The Surgeon General and the World Health Organization estimate that more than 700 U.S. infant deaths per year from SIDS are attributable to maternal smoking (CDC, 1991a, 1992b). However, this report concludes that at present there is not enough direct evidence supporting the contribution of ETS exposure to declare it a risk factor or to estimate its population impact on SIDS.

CONFERENCE HOSTS

CORPORATE PERSONNEL

Steve Parrish
General Counsel, PM USA
Vice President, External Affairs
Philip Morris U.S.A.
120 Park Avenue
New York, NY 10017
(212) 878-3074 Phone
(212) 878-2165 Facsimile

Overall responsibility for PM USA Legal, PM USA External Affairs and Corporate Scientific Affairs (ETS specific issue group.

Clare Purcell
Manager, Legal Issues
Philip Morris Management Corp.
P. O. Box 26583
Richmond, VA 23261
(804) 274-3656 Phone
(804) 274-5680 Facsimile

Responsible for identifying and coordinating legal issues relative to ETS activities with outside counsel. Contributes to Corporate Scientific Affairs planning and budgeting function and general project support.

Mary Pottorff
Manager, Corporate Scientific Affairs
Philip Morris Management Corp.
120 Park Avenue, 23rd Floor
New York, NY 10017
(212) 878-3899 Phone
(212) 907-5401 Facsimile

Responsible for monitoring, analyzing and developing strategic responses to environmental tobacco smoke (ETS) and indoor air issues in cooperation with corporate affairs staff. Initiates, directs and coordinates specific programs to address the issue from a corporate perspective.

Mayada Logue
Scientific and Regulatory Affairs Analyst
Philip Morris Management Corp.
P. O. Box 26583
Richmond, VA 23261
(804) 274-3189 Phone
(804 274-2921 Facsimile

Responsible for monitoring technical meetings and conferences for new developments related to ETS, air quality, risk assessment, health issues and environmental policy and regulation. Disseminates information for management decision-making and for use in formulating Corporate Affairs responses.

INTERNATIONAL PERSONNEL

Matthew Winokur
Director, Corporate Affairs
Philip Morris International
800 Westchester Avenue
Rye Brook, New York 10573-1301
(914) 335-9213 Phone
(914)335-9974 Facsimile

Overall responsibility for Corporate Affairs; ETS issue; monitoring, analyzing and developing strategic responses to public policy issues impacting Philip Morris International's business interests world-wide.

Anne Okoniewski
Coordinator, Research Analysis
Philip Morris International
800 Westchester Avenue
Rye Brook, New York 10573-1301
(914) 335-9275 Phone
(914) 335-9974 Facsimile

Special responsibility for research and analysis of the following issues: health, product integrity, ETS, social cost, general marketing issues, science, product liability, antis (individual, local and regional) and environmental issues.

USA PERSONNEL

Robert Pages
Director, Science and Technology
Philip Morris USA
P. O. Box 26583
Richmond, VA 23261
(804) 274-5405 Phone
(804) 274-2921 Facsimile

Responsible for the direction and coordination of scientific issues related to environmental tobacco smoke and indoor air quality worldwide.

Loreen McAlpin
Technical Analyst
Philip Morris USA
P. O. Box 26583
Richmond, VA 23261
(804) 274-2090 Phone
(804) 274-2921 Facsimile

Responsible for researching, analyzing, and disseminating information regarding environmental tobacco smoke and indoor air quality. Maintains databases of ETS/IAQ literature, IAQ meetings and research funding information.

EUROPEAN PERSONNEL

Helmut Reif
Director, Science & Technology
Philip Morris Europe
FTR Research & Development
Quai Jeanreanaud 3
CH-2003 Neuchâtel
Switzerland
(41) (38) 32 7628 Phone
(41) (38) 31 2476 Facsimile

Overall responsibility for the scientific and health-related back-up of ETS and IAQ related questions. Provides strategic direction to the scientific research in this field and input on proactive measures regarding ETS and IAQ.

Ruth Dempsey
Staff Scientist
Philip Morris Europe
Quai Jeanreanaud 3
CH-2003 Neuchâtel
Switzerland
(41) (38) 32 1111 Phone
(41) (38) 31 2476

Monitoring of scientific, health-related and regulatory questions. Preparation of targeted information and recommendations in the fields of smoking and health and indoor air quality.

LIST OF ATTENDEES
ETS WORLD CONFERENCE
MACKLOWE HOTEL
FEBRUARY 24 - 15, 1993

PHILIP MORRIS PERSONNEL

Bill Apple
Philip Morris USA
P. O. Box 26603
Richmond, VA 23261
Phone (804) 274-2801
Fax (804) 274-4572

Jim Boland
Philip Morris Washington Relations Office
Colorado Building
1341 G. Street, NW
Suite 900
Washington, DC 20005
Phone (202) 637-1508
Fax (202) 637-1505

John Boltz
Philip Morris USA
120 Park Avenue
New York, NY 10017
Phone (212) 880-4119
Fax (212) 907-5361

Tom Borelli
Philip Morris Corporate
120 Park Avenue
New York, NY 10017
Phone (212) 880-3874
Fax (212) 907-5464

Wendy Burrell
Philip Morris International
800 Westchester Avenue
Rye Brook, NY 10573
Phone (914) 335-9217
Fax (914) 335-9974

David Bushong
Philip Morris Corporate Services
166, Rue Joseph II
B-1040 Brussels
Belgium
Phone (32-2) 287-8011
Fax (32-2) 287-8048

Richard Carchman
Philip Morris USA
P. O. Box 26583
Richmond, VA 23261
Phone (804) 274-5794
Fax (804) 274-3982

Stig Carlson
Philip Morris EEMA
Philip Morris Europe, S.A.
Ave. De Cour 107
Case Postale
1001 Lausanne
Switzerland
Phone (41-21) 618-4184
Fax (41-21) 618-4618

Karen Chaikin
Philip Morris USA
Corporate Affairs
120 Park Avenue
New York, NY 10017
Phone (212) 880-3358
Fax (212) 907-5716

David Davies
Philip Morris Management Corp.
120 Park Avenue
New York, NY 10017
Phone (212) 880-3203
Fax (212) 907-5344

Ruth Dempsey
Philip Morris Europe
3 Quai Jeanrenaud
CH-2003 Neuchâtel
Switzerland
Phone (41) (38) 32 1111
Fax (41) (38) 31 2476

Darienne Dennis
Philip Morris International
800 Westchester Avenue
Rye Brook, NY 10573-1301
Phone (914) 335-9223
Fax (914) 335-9974

PHILLIP MORRIS PERSONNEL

Phil Francis
Philip Morris Australia
Philip Morris Ltd.
252 Chesterville Road
Moorabbin, Victoria 3189
Australia
Phone (61-3) 556-0210
Fax (61-3) 555-2686

Colin Goddard
Philip Morris Asia, Inc.
23rd Floor
Two Pacific Place
88 Queensway
HONG KONG
Phone (85-2) 825-1553
Fax (85-2) 524-6394

Aurora Marina Gonzalez
PM Latin America
800 Westchester Avenue
Rye Brook, NY 10573-1301
Phone (914) 335-7350
Fax (914) 335-7928

Jan Goodheart
Philip Morris K.K.
Akasaka Twin Tower
17-22, Akasaka 2-chome
Minato-Ku, Tokyo 107
JAPAN
Phone (81-3) 5562-7295
Fax (81-3) 5562-9789

Vic Han
Philip Morris USA
120 Park Avenue
New York, NY 10017
Phone (212) 878-2796
Fax (212) 907-5361

Henrick Hansen
Philip Morris Corporate Services
166, Rue Joseph II
B-1040 Brussels
Belgium
Phone (32-2) 287-8068
Fax (32-2) 287-8069

Judy Hargrave
Philip Morris Australia
Philip Morris Ltd.
252 Chesterville Road
Moorabbin, Victoria 3189
Phone (61-3) 556-0242
Fax (61-3) 532-2289

Robert Kaplan
Philip Morris International
800 Westchester Avenue
Rye Brook, NY 10573-1301
Phone (914) 335-9216
Fax (914) 335-9974

Denise Keane
Philip Morris Management Corp.
120 Park Avenue
New York, NY 10017
Phone (212) 880-3076
Fax (212) 907-5401

Ted Lattanzio
Philip Morris USA
120 Park Avenue
New York, NY 10017
Phone (212) 880-3851
Fax (212) 907-5716

Mayada Logue
Philip Morris Management Corp.
P. O. Box 26583
Richmond, VA 23261
Phone (804) 274-3189
Fax (804) 274-2921

Loreen McAlpin
Philip Morris USA
P. O. Box 26583
Richmond, VA 23261
Phone (804) 274-2090
Fax (804) 274-2921

Ellen Merlo
Philip Morris USA
120 Park Avenue
New York, NY 10017
Phone (212) 880-3230
Fax (212) 878-2165

Dave Merrill
Philip Morris USA
P. O. Box 26603
Richmond, VA 23261
Phone (804) 274-2954
Fax (804) 274-4572

2024103533

PHILIP MORRIS PERSONNEL

Eva Montgomery
Philip Morris EEMA
Philip Morris Europe, S.A.
Ave. De Cour 107
Case Postale
1001 Lausanne
Switzerland
Phone (41-21) 618-4184
Fax (41-21) 618-4618Marie Claire Nas

Philip Morris EEMA
Philip Morris Europe, S.A.
Ave. De Cour 107
Case Postale
1001 Lausanne
Switzerland
Phone (41-21) 618-4292
Fax (41-21) 618-4618

Anne Okoniewski
Philip Morris International
800 Westchester Avenue
Rye Brook, NY 10573-1301
Phone (914) 335-9275
Fax (914) 335-9974

Robert Pages
Philip Morris USA
P. O. Box 26583
Richmond, VA 23261
Phone (804) 274-5405
Fax (804) 274-2921

Steve Parrish
Philip Morris Management Corp.
120 Park Avenue
New York, NY 10017
Phone (212) 880-3074
Fax (212) 878-2165

Mary Pottorff
Philip Morris Management Corp
120 Park Avenue
New York, NY 10017
Phone (212) 880-3899
Fax (212) 907-5401

Lance Pressl
Philip Morris USA
120 Park Avenue
New York, NY 10017
Phone (212) 880-4131
Fax (212) 907-5315

Clare Purcell
Philip Morris Management Corp.
P. O. Box 26583
Richmond, VA 23261
Phone (804) 274-3656
Fax (804) 274-4780

Helmut Reif
Philip Morris Europe
3 Quai Jeanrenaud
CH-2003 Neuchâtel
Switzerland
Phone (41) (38) 32 7628
Fax (41) (38) 31 2476

Cesar Rodriguez
Philip Morris Latin America
800 Westchester Avenue
Rye Brook, NY 10573-1301
Phone (914) 335-7349
Fax (914) 335-7928

James Spector
Philip Morris USA
120 Park Avenue
New York, NY 10017
Phone (212) 880-3585
Fax (212) 808-5236

William Taylor
Philip Morris USA
P. O. Box 26603
Richmond, VA 23261
Phone (804) 274-4413
Fax (804) 274-5341

Tina Walls
Philip Morris USA
120 Park Avenue
New York, NY 10017
Phone (212) 878-2302
Fax (212) 907-5716

Matthew Winokur
Philip Morris International
800 Westchester Avenue
Rye Brook, NY 10573-1301
Phone (914) 335-9213
Fax (914) 335-9974

Gerard Wirz
Philip Morris Corporate Services
166, Rue Joseph II
B-1040 Brussels
Belgium
Phone (32-2) 287-8011
Fax (32-2) 287-8069

INVITED SPEAKERS

COLUMBIA UNIVERSITY

Vincent T. Covello
School of Public Health
60 Haven Avenue, B-1, Room 106
New York, NY 10032
Phone (212) 305-3464
Fax (212) 749-3590

Susan Santos
Center for Risk Communication
School of Public Health
68 Crestview Drive
Malden, MA 02148
Phone/Fax (617) 322-1923

COVINGTON & BURLING

Charles Lister
Acheson House
46 Hertford Street
London, W1Y 7TF
United Kingdom
Phone (44-71) 495-5655
Fax (44-71) 495-3101

Melinda Sidak
Covington & Burling
1201 Pennsylvania avenue
P. O. Box 7566
Washington, DC 20004
Phone (202) 662-5444
Fax (202) 662-6291

CONSTANGY, BROOKS & SMITH

Pat Tyson
Suite 2400
230 Peachtree St., NW
Atlanta, GA 30303
Phone (404) 525-8622
Fax (404) 525-6955

HEALTHY BUILDINGS INTERNATIONAL, INC.

Gray Robertson
10378 Democracy Lane
Fairfax, VA 22030
Phone (703) 352-0102
Fax (703) 352-0151

SHOOK, HARDY & BACON

Tony Andrade
PM Europe
Brillancourt 5, Case Postale
1001 Lausanne, Switzerland
Phone (41-21) 618-6495
Fax (41-21) 617-6910

Bill Colby
Shook, Hardy & Bacon Law Offices
One Kansas City Place
1200 Main Street
Kansas City, MO 64105
Phone (816) 474-6550
Fax (816) 421-5547

Greg Fowler
Philip Morris Australia
Philip Morris Ltd.
252 Chesterville Road
Moorabbin, Victoria 3189
Australia
Phone (61-3) 556-0251
Fax (61-3) 532-2289

THE WIRTHLIN GROUP

Dave Richardson
420 Lexington Avenue
11th Floor
New York, NY 10017
Phone (212) 210-8700
Fax (212) 661-7632

INVITED GUESTS

APCO

Margery Kraus
1155 21st Street, NW
Suite 1000
Washington, DC 20036
Phone (202) 778-1010
Fax (202) 861-0525

Tom Hackaday
1155 21st Street, NW
Suite 1000
Washington, DC 20036
Phone (202) 778-1036
Fax (202) 861-0525

CONSTANGY, BROOKS & SMITH

Neil Wasser
Suite 2400
230 Peachtree St., NW
Atlanta, GA 30303
Phone (404) 525-8622
Fax (404) 525-6955

SHOOK, HARDY & BACON

Dennis Neutze
One Kansas City Place
1200 Main Street
Kansas City, MO 64105
Phone (816) 474-6550
Fax (816) 421-5547

2024103536

SUPPORT STAFF

VIDEO SERVICES

Ray Cahen
Philip Morris USA
Technical Training Center
P. O. Box 26603
Richmond, VA 23261
Phone (804) 274-2264
Fax (804) 274-2758

Jim Mohr
Philip Morris USA
Technical Training Center
P. O. Box 26603
Richmond, VA 23261
Phone (804) 274-2943
Fax (804) 274-2758

Charlie Owens
Philip Morris USA
Technical Training Center
P. O. Box 26603
Richmond, VA 23261
Phone (804) 274-5967
Fax (804) 274-2758

Dru Stevens
Philip Morris USA
Technical Training Center
P. O. Box 26603
Richmond, VA 23261
Phone (804) 274-7893
Fax (804) 274-2758

SECRETARIAL SERVICES

Gloria Charlery
Philip Morris Management Corp.
120 Park Avenue
New York, NY 10017
Phone (212) 880-3197
Fax (212) 907-5344

Anis Buonpensiere
Philip Morris International
800 Westchester Avenue
Rye Brook, NY 10573-1301
Phone (914) 335-1295
Fax (914) 335-9974

ETS WORLD CONFERENCE
MACKLOWE HOTEL AND CONFERENCE CENTER
NEW YORK, NEW YORK
FEBRUARY 24 - 25, 1993

Welcome to the Macklowe Hotel and Conference Center. This folder contains the information you will need for the ETS World Conference. It includes the final agenda, your risk communication exercise assignment for Day 2, speaker biographies, background ETS Material, information regarding the available secretarial services, and other general information to make your stay more comfortable.

If you need any further information, please feel free to contact Clare Purcell, Anne Okoniewski or Loreen McAlpin.

2024103538

SECRETARIAL SERVICES

Anis Buonpensiere and Gloria Charlery will be providing secretarial support during the conference from 8:00 am - 6:00 pm in Room 507 during the Conference.

Phone numbers where messages can be left:

(212) 789-7740
(212) 789-7710

Fax number:

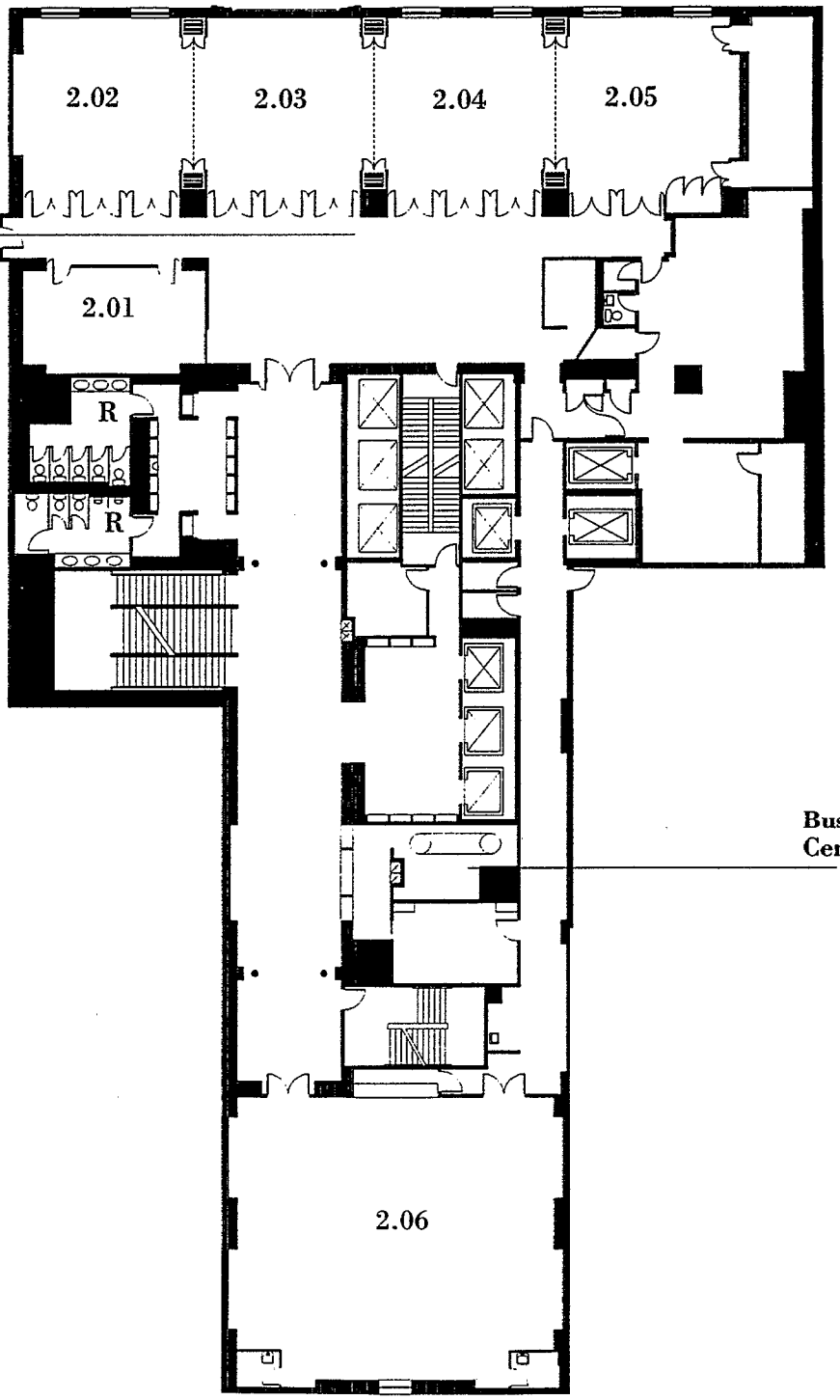
(212) 789-7740

If you need to use the office after 6:00 pm, please contact Loreen McAlpin.

Second Floor

Hotel Macklowe
Macklowe Conferen Cente
212/768.4400

45 West 44th Street
New York, New York 10036
Fax: 212/789.7698

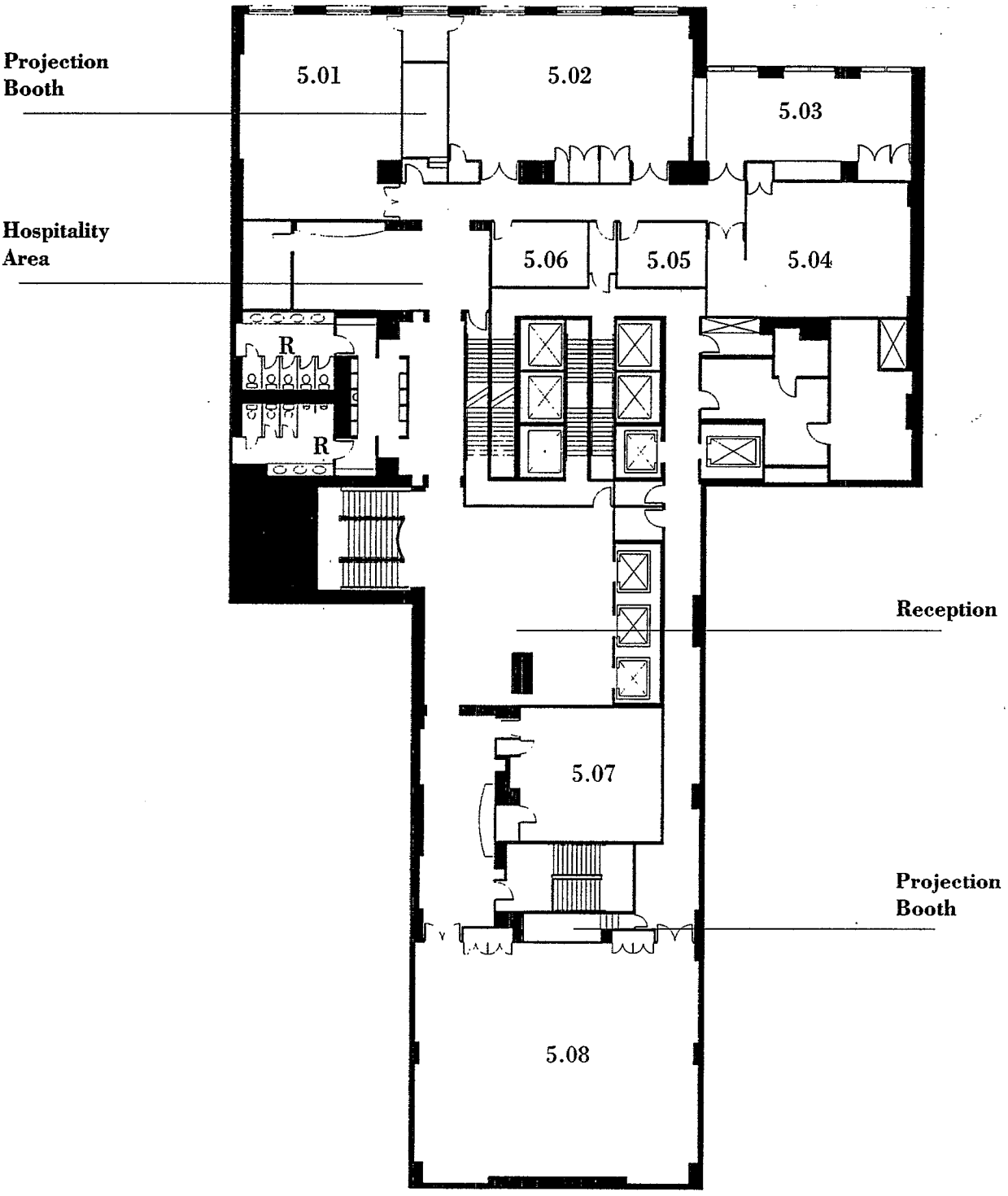


20241103540

Fifth Floor

Hotel Macklowe
Macklowe Conference Center
212/768.4400

145 West 44th Street
New York, New York 10036
Fax: 212/789.7698



20241035 41

ROMAN CATHOLIC CHURCHES
ASH WEDNESDAY SERVICES
WEDNESDAY, FEBRUARY 24, 1993

The Church of St. Agnes
158 E. 44th Street (Between 3rd & Lexington)

Ashes will be given out continuously in the school's auditorium between 7:00 am - 6:00 pm.

The Daily News Building
220 E. 42nd Street (Between 2nd & 3rd Avenues)

Ashes will be given out after each mass:

7:30 am
8:00 am
8:30 am
12:05 pm
12:35 pm
1:05 pm
5:15 pm

St. Malachy's
West 49th Street - between Broadway and 8th Avenue

Ashes will be given out after each mass:

8:00 am
12:15 pm
1:15 pm
5:15 pm

RISK COMMUNICATION EXERCISE
GROUP ASSIGNMENT

GROUP I Participants

Facilitator: Ellen Merlo

1. Tony Andrade
2. David Bushong
3. Karen Chaikin
4. Ruth Dempsey
5. Greg Fowler
6. Colin Goddard
7. Dave Merrill
8. Anne Okoniewski
9. Bob Pages
10. Neil Wasser

GROUP II Participants

Facilitator: Clare Purcell

1. Bill Apple
2. Tom Borelli
3. Stig Carlson
4. David Davies
5. Vic Han
6. Loreen McAlpin
7. Cesar Rodriguez
8. Jim Spector
9. Pat Tyson
10. Tina Walls

GROUP III Participants

Facilitator: Mayada Logue

1. Wendy Burrell
2. Richard Carchman
3. Darienne Dennis
4. Phil Francis
5. Jan Goodheart
6. Tom Hockaday
7. Bobby Kaplan
8. Denise Keane
9. Eva Montgomery
10. Lance Pressl
11. Gerard Wirz

GROUP IV Participants

Facilitator: Matt Winokur

1. John Boltz
2. Aurora Gonzalez
3. Henrick Hansen
4. Judith Hargrave
5. Margery Kraus
6. Ted Lattanzio
7. Marie-Clare Nas
8. Dennis Neutze
9. Steve Parrish
10. Mary Pottorff
11. Helmut Reif

*** PLEASE PICK UP YOUR GROUP'S BACK-UP MATERIALS FOR THIS EXERCISE WEDNESDAY IN THE BUSINESS OFFICE.**

2024103543